UNITED STATES DISTRICT COURT

EASTERN DISTRICT OF WASHINGTON

IN RE HANFORD NUCLEAR RESERVATION LITIGATION	MASTER CASE FILE NO. CY-91-3015-AAM
	ORDER RE SUMMARY JUDGMENT

This document relates to: <u>All cases</u>

ORDER RE SUMMARY JUDGMENT-   1

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18
19
20
21
22
23
24
25
26
27
28

TABLE OF CONTENTS

ORDER RE SUMMARY JUDGMENT-   2

I.   INTRODUCTION

BEFORE THE COURT are the defendants' motions for summary judgment (Ct. Rec. 902, 904, 930, 932 and 933), accompanying motions in limine (Ct. Rec. 902, 906, 907 and 1007), and various other assorted motions which will be identified in the text of this order.

Extensive oral argument was heard on December 2, 1997 regarding the evidentiary standard for evaluating plaintiffs' claims on summary judgment. As will be apparent, resolution of this pivotal threshold issue affects resolution of the various motions in limine. The plaintiffs have requested additional oral argument on defendants' summary judgment motions and motions in limine. (Ct. Rec. 1193).

LR 7(h)(2) states that parties may request oral argument in support of or in opposition to any motion and that without such a request, oral argument is waived. Notwithstanding this procedure, LR 7(h)(3) provides that the court may, in its discretion, determine oral argument is not warranted and proceed to determine any motion brought under this rule without oral presentation.

Pursuant to LR 7(h)(3), this court finds additional oral argument is not warranted. Oral argument was heard on the singular most important and overarching issue in this case, that being the quantum of proof necessary for plaintiffs to survive summary judgment. Based on that oral argument, the court believes it fully understands the position of the parties

ORDER RE SUMMARY JUDGMENT-   3

1 regarding the requisite quantum of proof. Additional oral

2 argument, particularly on the motions in limine[1], would not be

3 beneficial to the court and indeed, may well add unnecessarily to

4 the complexity of those motions. The extensive written

5 submissions of the parties provide the court with all of the

6 information it needs to understand their respective arguments.

7 The court believes such understanding is manifested in this

8 order.

9   For all of these reasons, plaintiffs' requests and motion

10 for oral argument (Ct. Rec. 1193) are **DENIED**.[2]

11

12 **II. BACKGROUND**

13   This litigation represents the consolidation of separate

14 lawsuits filed by various groups of plaintiffs starting in 1990.

15 In February 1991, five separate actions were consolidated:

16 Sandra Evenson, et al. v. United States Environmental Protection

17 Agency, et al., CY-90-3067-AAM; Kathryn Hamilton, et al., v. E.I.

18 DuPont De Nemours and Company, et al., CY-90-3069-AAM; Kenneth

19 Wahpat, et al., v. General Electric Co., et al., CY-90-3091-AAM;

20 E.S. Criswell, et al., v. E.I. DuPont De Nemours, et al., CY-90-

21 _____

22   [1] The parties agreed that evidentiary hearings on the
    motions in limine were unnecessary.

23

24   [2] This case is distinguishable from Jasinski v. Showboat
    Operating Co., 644 F.2d 1277 (9th Cir. 1981). First, this court
    has complied with its own local rules. Secondly, oral argument

25 was entertained to an extent the court believes was warranted.
    Thirdly, the court believes its resolution of the issues shows an

26 understanding of the parties' arguments which would not have been
    enhanced by additional oral argument. No prejudice results from

27 the lack of additional oral argument.

28 **ORDER RE SUMMARY JUDGMENT-   4**

1 3106-AAM; and <u>Jaros, et al., v. E.I. DuPont De Nemours, et al.</u>,

2 CY-90-3107-AAM. (Ct. Rec. 1). A joint consolidated complaint

3 was subsequently filed by these plaintiffs. (Ct. Rec. 15).

4   The <u>Wahpat</u> plaintiffs were dismissed from the consolidated

5 litigation by separate order of this court issued in February

6 1995. (Ct. Rec. 526). As this consolidated litigation has

7 proceeded over the years, two primary plaintiff groups have

8 emerged: 1) the <u>Evenson</u> group represented by Tom Foulds, Esq.,

9 and Associates in Seattle, Washington; and 2) the

10 <u>Jaros/Hamilton/Criswell</u> group collectively represented by Roy

11 Haber, Esq., Eugene, Oregon; Berger & Montague, Philadelphia,

12 Pennsylvania; and Waite, Schneider, Bayless & Chesley Co.,

13 Cincinnati, Ohio. These are the two largest groups of

14 plaintiffs.

15   Subsequent to the court's February 1991 order of

16 consolidation, several additional cases were joined to the <u>In re</u>

17 <u>Hanford</u> consolidated litigation, including: <u>Roseman, et al. v.</u>

18 <u>General Electric Co., et al.</u>, CY-91-3045-AAM; <u>Seaman, et al. v.</u>

19 <u>E.I. DuPont De Nemours, et al.</u>, CY-91-3080-AAM; <u>Miller v. E. I.</u>

20 <u>DuPont De Nemours, et al.</u>, CY-92-3069-AAM; <u>Durfey, et al., v.</u>

21 <u>E.I. DuPont De Nemours, et al.</u>, CY-93-3087-AAM; and <u>Thomson, et</u>

22 <u>al. v. E.I. DuPont De Nemours, et al.</u>, CY-94-3067-AAM.[3]

23   Separate litigation before this court involving claims

24 _____

25   [3] <u>Thomson</u> and <u>Durfey</u> involve claims for medical monitoring
   relief which are not specifically before the court on the current
   summary judgment motions. Nevertheless, resolution of the

26 current summary judgment motions will almost certainly have some
   bearing on the future of medical monitoring claims.

27

28 **ORDER RE SUMMARY JUDGMENT-   5**

1   similar to those found in the In re Hanford consolidated

2   litigation includes: 1)   Berg, et al., v. E. I. DuPont De

3   Nemours, et al., CY-96-3151-AAM; and 2) Jim, et al., v. E.I.

4   DuPont De Nemours, et al., CY-97-3061-AAM.   The claims of several

5   plaintiffs, formerly of the Berg group, have been consolidated

6   with this litigation.

7   The approximately 3,000 plaintiffs in this consolidated

8   litigation allege they have suffered personal injury or will

9   suffer future injury as a result of exposure to radioactive and

10   non-radioactive emissions from the Hanford Nuclear Reservation

11   located in southeastern Washington.   They seek damages for

12   present injuries including thyroid cancer, non-neoplastic thyroid

13   diseases, and various non-thyroid cancers.   They also seek

14   damages based on the prospect of future injuries.

15   Pursuant to contract with the United States Department of

16   Energy (DOE) and its predecessors, the defendants- E.I. DuPont De

17   Nemours and Company ("DuPont"), General Electric Company ("GE"),

18   UNC Nuclear Industries, Inc. ("UNC"), Atlantic Richfield Company

19   ("ARCO"), and Rockwell International Corporation ("Rockwell")-

20   operated the Hanford Nuclear Reservation ("Hanford") at various

21   times from approximately 1943 to 1987.

22   For most of that period, the function of Hanford was to

23   produce plutonium for use in nuclear weapons.   In addition to

24   plutonium (Pu-239), other radionuclides were created in the

25   plutonium production process, including iodine-131 (I-131 or

26   radioiodine), strontium-90 (Sr-90), ruthenium-103 (Ru-103),

27   ruthenium-106 (Ru-106), cerium-144 (Ce-144) and cesium-137 (Cs-

28   **ORDER RE SUMMARY JUDGMENT-   6**

1 137).   The radioactive emissions chiefly at issue in this

2 litigation are I-131 and Pu-239.

3   This consolidated litigation has been divided into phases.

4 Phase I dealt with interrogatory and document discovery by both

5 plaintiffs and defendants.   During Phase II, the parties were to

6 focus on the issue of causation through preparation of expert

7 reports and the conducting of expert discovery.   Phase II has

8 come to be known as the "generic" causation phase.[4] Defendants'

9 summary judgment motions follow the completion of Phase II.   Upon

10 resolution of these motions, remaining claims will proceed into

11 Phase III which will cover individual causation discovery,

12 liability and any other remaining issues.

13   This court has always considered causation to be the pivotal

14 issue and therefore, opted to address it before addressing

15 liability (breach of duty).   Defendants' summary judgment motions

16 seek dismissal of a majority of plaintiffs' claims on the basis

17 that their alleged health conditions cannot be linked to Hanford

18 emissions.

19

20 **III.   SUMMARY JUDGMENT STANDARD**

21   The purpose of summary judgment is to avoid unnecessary

22 trials when there is no dispute as to the facts before the court.

23 _____

24   [4] In its October 3, 1995 order re Phase II Schedule (Ct.
Rec. 575), this court stated that if plaintiffs were unable to
establish **general** principles of causation after discovery was

25 completed regarding all relevant radionuclides and time periods,
then "presumably their claims would be subject to successful

26 challenge through dispositive motions" and "such a result would
obviate the need to conduct discovery on an individual-by-

27 individual basis."

28 **ORDER RE SUMMARY JUDGMENT-   7**

1 <u>Zweig v. Hearst Corp.</u>, 521 F.2d 1129 (9th Cir.), <u>cert. denied</u>,

2 423 U.S. 1025 (1975).   Under Fed. R. Civ. Proc. 56, a party is

3 entitled to summary judgment where the documentary evidence

4 produced by the parties permits only one conclusion. <u>Anderson v.</u>

5 <u>Liberty Lobby, Inc.</u>, 477 U.S. 242, 106 (1986); <u>Semegen v.</u>

6 <u>Weidner</u>, 780 F.2d 727 (9th Cir. 1985).   Summary judgment is

7 precluded if there exists a genuine dispute over a fact that

8 might affect the outcome of the suit under the governing law.

9 <u>Anderson</u>, 477 U.S. at 248.

10 The moving party has the initial burden to prove that no

11 genuine issue of material fact exists. <u>Matsushita Elec.</u>

12 <u>Industrial Co. v. Zenith Radio Corp.</u>, 475 U.S. 574, 586 (1986).

13 Once the moving party has carried its burden under Rule 56, "its

14 opponent must do more than simply show that there is some

15 metaphysical doubt as to the material facts." <u>Id</u>. The party

16 opposing summary judgment must go beyond the pleadings to

17 designate specific facts establishing a genuine issue for trial.

18 <u>Celotex Corp. v. Catrett</u>, 477 U.S. 317, 325 (1986).

19 In ruling on a motion for summary judgment, all inferences

20 drawn from the underlying facts must be viewed in the light most

21 favorable to the nonmovant. <u>Matsushita</u>, 475 U.S. at 587.

22 Nonetheless, summary judgment is required against a party who

23 fails to make a showing sufficient to establish an essential

24 element of a claim, even if there are genuine factual disputes

25 regarding other elements of the claim. <u>Celotex</u>, 477 U.S. at 322-

26 23.

27 //

28 **ORDER RE SUMMARY JUDGMENT-   8**

IV.   BURDEN OF PRODUCTION/BURDEN OF PROOF

A.   **Sufficiency of Proof/Generic Causation Stage**

Plaintiffs and defendants have fundamentally different views on the nature of plaintiffs' burden at this "generic" causation stage of the proceedings. Plaintiffs contend their burden is to prove only that Hanford emissions are "capable of causing" the various health conditions claimed by them and that they were exposed to Hanford emissions in dose ranges "capable of causing" those conditions. So long as this burden is met, plaintiffs contend they are entitled to have their claims considered by a jury.[5]

Defendants contend plaintiffs' burden is to establish the dose at which their risk of each claimed disease is doubled. Unless exposed to such a "doubling dose," defendants assert an inference cannot arise that exposure was a "more likely than not" cause of the particular disease and therefore, the claim cannot be considered by a jury.

"Generic causation," as that term is **commonly** used in the caselaw, asks whether an agent is **capable** of causing a particular disease. <u>Hilao v. Estate of Marcos</u>, 103 F.3d 767, 788 (9th Cir. 1996) (Rymer, J. concurring in part and dissenting in part) ("contrasting generic causation-that the defendant was responsible for a tort which had the capacity to cause the harm alleged-with individual proximate cause and individual damage").

---

[5] At various points in their written submissions, plaintiffs contend meeting their "generic" causation burden does not even require them to offer any proof of the dose level to which they were potentially exposed.

ORDER RE SUMMARY JUDGMENT-   9

1   Defendants concede radiation exposure is "capable of

2 causing" certain of the conditions at issue, notably thyroid

3 cancer and non-autoimmune hypothyroidism. However, they dispute

4 what plaintiffs assert are the dose levels at which radiation is

5 "capable of causing" those conditions. As to certain other

6 conditions, including various types of non-neoplastic thyroid

7 disease, defendants contend plaintiffs' expert evidence is

8 inadmissible and insufficient to raise a genuine issue of

9 material fact that radiation exposure is even "capable of

10 causing" the condition. Obviously, if radiation exposure is not

11 "capable of causing" a particular condition, any claims based on

12 that condition cannot survive summary judgment.

13   However, even if radiation exposure is "capable of causing"

14 a particular condition, that alone does **not** allow the claim to be

15 considered by a jury. Washington tort law applies to personal

16 injury claims brought under the Price-Anderson Act. In re

17 Hanford, 780 F. Supp. 1551, 1570 (E.D. Wash. 1991). Under

18 Washington tort law, a plaintiff must show the "act complained of

19 'probably' or 'more likely than not' caused the subsequent

20 disability." Schudel v. General Electric Co., 120 F.3d 991, 996

21 (9th Cir. 1997), cert. denied 118 S.Ct. 1560 (1998), quoting

22 O'Donoghue v. Riggs, 73 Wn. 2d 814, 440 P.2d 823, 830 (1968).

23 Evidence that radiation is "capable of causing" the injury raises

24 only a "possibility" it is in fact a cause of the injury. Such

25 evidence invites a jury to speculate whether radiation exposure

26 is in fact a cause of the injury and, **by itself**, is of no

27 assistance to a jury. Id. citing Daubert v. Merrell Dow

28 **ORDER RE SUMMARY JUDGMENT-**   **10**

Pharmaceuticals, Inc. (aka "Daubert II"), 43 F.3d 1311, 1320-22

(9th Cir. 1995).[6]

In Schudel, the Ninth Circuit commented that the Washington

tort law standard was "virtually the same" as the California tort

law standard applied in Daubert II. In Daubert II, two minors

brought suit claiming they suffered limb reduction birth defects

because their mothers had taken a drug called Bendectin. Under

California tort law, plaintiffs were required to show "not merely

that Bendectin increased the likelihood of injury, but that it

more likely than not caused **their** injuries." In terms of

statistical proof, this required plaintiffs to show their

mothers' ingestion of Bendectin "doubled" the likelihood of birth

defects: "Because the background rate of limb reduction defects

is one per thousand births, plaintiffs must show that among

children of mothers who took Bendectin the incidence of such

defects was more than two per thousand." 43 F.3d at 1320.

As it turned out, none of the plaintiffs' epidemiological

experts claimed the ingestion of Bendectin during pregnancy more

than doubled the risk of birth defects. None of them stated that

children whose mothers took Bendectin were more than twice as

likely to develop limb reduction birth defects as children whose

mothers did not. In epidemiological terms, this meant none of

the expert studies opined the relative risk was greater than

---

[6] See also Ambrosini v. Labarraque, 101 F.3d 129, 135-36
(D.C. Cir. 1996).

ORDER RE SUMMARY JUDGMENT-   11

1 2.0.[7] As such, none of these studies showed causation under

2 California's preponderance standard.   The circuit found the

3 studies would not be helpful, but would only serve to confuse the

4 jury if offered to prove, rather than refute causation.

5 According to the circuit: "A relative risk of less than two may

6 suggest teratogenicity, but it actually tends to disprove legal

7 causation, as it shows that Bendectin does not double the

8 likelihood of birth defects." Id. at 1321 (emphasis in text).

9   Clearly, under Schudel, plaintiffs' evidence that radiation

10 is "capable of causing" their injuries at certain dose ranges is

11 insufficient, by itself, to get their claims before a jury.

12 Therefore, the question is where exactly does such evidence get

13

——————————————————

14   [7] According to the Federal Judicial Center, Reference
Manual on Scientific Evidence, "Reference Guide on Epidemiology,"
15 (1994) at pp. 168-69:

16   The civil burden of proof is described most
            often as requiring the fact finder to 'believe
17   that what is sought to be proved . . . is more
            likely true than not true.' The relative risk
18   from an epidemiological study can be adapted to
            this 50% plus standard to yield a probability or
19   likelihood that an agent caused an individual's
            disease. The threshold for concluding that an
20   agent was more likely the cause of a disease than
            not is a relative risk greater than 2.0. Recall that
21   a relative risk of 1.0 means that the agent has
            no effect on the incidence of the disease. When
22   the relative risk reaches 2.0, the agent is responsible
            for an equal number of cases of disease as all
23   other background causes. Thus, a relative risk
            of 2.0 implies a 50% likelihood that an exposed
24   individual's disease was caused by the agent. A
            relative risk greater than 2.0 would permit an
25   inference that an individual plaintiff's disease
            was more likely than not caused by the implicated
26   agent.

27

28 **ORDER RE SUMMARY JUDGMENT-   12**

1 | the plaintiffs, presuming of course the evidence is admissible.

2 | Plaintiffs contend defendants' "doubling of risk" standard

3 | pertains, **if at all**, to what this court has referred to as Phase

4 | III when individual causation matters are to be taken up.

5 | Indeed, plaintiffs argue "doubling of risk" and the "more likely

6 | than not" evidentiary standard from which it derives, is entirely

7 | irrelevant because they need only prove to a jury's satisfaction

8 | that their radiation exposure was a "substantial factor" in

9 | causing their injuries.

10 | The current state of scientific knowledge does not allow the

11 | plaintiffs to directly prove that radiation exposure, and

12 | specifically Hanford emissions, caused their asserted health

13 | conditions. These health conditions- thyroid cancer, non-

14 | neoplastic thyroid disease, and various non-thyroid cancers-

15 | occur regularly in the unexposed population for any number of

16 | reasons (i.e. diet, smoking, genetic defect, etc.). Plaintiffs'

17 | experts concede as much.

18 | With regard to cancer, Baruch Modan, an epidemiologist,

19 | says:

20 |
> Radiation-induced cancer has no unique
> characteristics in terms of tissue or cell

21 |
> type; there is no way to prove which patients
> developed cancer due to the radiation treatment

22 |
> and which ones would have developed it anyway.
> In other words, we cannot predict which individuals

23 |
> in the irradiated population will develop cancer,
> nor can we confirm that that cancer developed in

24 |
> a specific individual because of the irradiation.

25 | Modan, "Low Dose Radiation Carcinogenesis- Issues and

26 | Interpretation: The 1993 G. William Morgan Lecture," 65 **Health**

27 | **Physics** 475 (Nov. 1993), at p. 478.

28 | **ORDER RE SUMMARY JUDGMENT-   13**

1   The same is true with regard to non-neoplastic thyroid

2 disease as confirmed by Edward Radford, M.D., also an

3 epidemiologist. At his deposition, Radford acknowledged that a

4 medical doctor cannot determine whether such disease was caused

5 by radiation exposure merely because of the fact of exposure.

6 (Radford Dep. at pp. 356-358). According to Radford, "that is

7 why you use epidemiologic methods to collect these cases and look

8 at them systematically." (Id. at p. 358).

9   As in Daubert II, plaintiffs here are forced to rely on

10 experts who present circumstantial proof of causation, in

11 particular, epidemiological proof. Epidemiologists use the

12 statistical measure of "relative risk" to indicate the strength

13 of association between exposure and disease. As Daubert II

14 points out, the "relative risk" determines whether there is a

15 "doubling of risk." Statistical proof is sufficient to get a

16 claim before a jury only if it shows a "doubling of risk" between

17 exposure and the condition. In cases where statistical proof

18 must be resorted to, such proof meets the "more likely than not"

19 sufficiency standard only if a "doubling of risk" is shown.

20   Plaintiffs contend their case does not boil down to

21 epidemiological proof and that Daubert II is distinguishable from

22 the instant case. In Daubert II, the Ninth Circuit observed that

23 scientists did not know **how** teratogens (chemicals known to cause

24 limb reduction birth defects) cause their damage, and that the

25 biological chain of events that leads from an expectant mother's

26 ingestion of a teratogenic substance to the stunted development

27 of a baby's limbs could not be reconstructed. In the instant

28 **ORDER RE SUMMARY JUDGMENT-   14**

1 case, plaintiffs assert the scientific community understands **how**

2 radiation causes its damage. According to plaintiffs, this

3 understanding is derived from, among other things, observation of

4 tumor growth in animals exposed to radiation.

5   While it may be true there is greater scientific

6 understanding of the biological mechanism by which radiation

7 induces cancer as opposed to the biological mechanism by which

8 teratogens cause birth defects, the fact remains, as acknowledged

9 by plaintiffs and their experts, that radiation-induced cancer

10 and disease cannot be distinguished from cancer and disease

11 induced by any of the other myriad of potential causes.[8] Actual

12 cause cannot be determined.

13   During oral argument, plaintiffs' counsel conceded that a

14 point is never reached where it can be said that an individual's

15 cancer was caused by radiation, and specifically by Hanford

16 releases. Asked whether a physician put on the stand could

17 testify to a reasonable medical certainty that his patient's

18 condition was caused by radiation, counsel conceded not so in the

19 absence of epidemiological proof. Counsel suggested thyroid

20 cancer might present an exception because of epidemiological

21 evidence that it is the only established environmental cause.

22 _____

          [8] Plaintiffs' counsel are wrong in suggesting <u>Daubert II</u>
23 was a "no evidence" case. In <u>Daubert II</u>, the plaintiffs'
   experts, **in addition** to testifying that statistical studies
24 showed an increase in the risk of birth defects, testified
   Bendectin is a teratogen because it causes birth defects when
25 tested on animals and because it is similar in chemical structure
   to other suspected teratogens. However, all of this evidence at
26 best showed Bendectin was "capable of causing" birth defects and
   this was insufficient to get the case before a jury. <u>Daubert II</u>,
27 43 F.3d at 1320-22.

28 **ORDER RE SUMMARY JUDGMENT-   15**

However, even here, counsel acknowledged the existence of non-environmental causes and the fact that Hanford releases would not constitute the only potential type of radiation exposure.

That epidemiological proof is vital to plaintiffs' case is manifested in their expert evidence which is discussed in detail _infra_. It must be recognized that epidemiology addresses **whether an agent can cause a disease and not whether an agent did cause a particular plaintiff's disease.** Federal Judicial Center Reference Manual on Scientific Evidence, "Reference Guide on Epidemiology," (1994) at p. 167.[9] In other words, epidemiology can answer the generic causation inquiry of whether an agent is capable of causing a disease, but it cannot answer the question of whether the agent caused the disease in a specific individual.

In determining whether the association between an agent and a disease is causal (i.e. whether the agent can cause the disease), an epidemiologist considers a number of different factors including: 1) strength of the association; 2) temporal relationship; 3) consistency of the association; 4) biological plausibility; 5) consideration of alternative explanations; 6) specificity of the association; and 7) dose-response relationship. These are known as Koch's postulates. _Id_. at p. 161.[10]

"Relative risk" measures the factor known as "strength of

---

[9] Hereinafter, "Reference Guide on Epidemiology."

[10] Plaintiffs' and defendants' refer to a variation of these known as Hill's postulates which includes: 1) strength and consistency of association; 2) dose-response relationship; 3) experimental evidence; 4) plausibility; and 5) coherence.

ORDER RE SUMMARY JUDGMENT-   16

1 the association." It is one of the "cornerstones" of causal

2 inference. The higher the relative risk, the greater the

3 likelihood the relationship is causal. Id.

4   "Consistency of the association" is "measured by comparing

5 the association between the purported cause and effect identified

6 in one study with the results of other studies and relevant

7 scientific knowledge." Joint Eastern & Southern Dist. Asbestos

8 Litigation, 52 F.3d 1124, 1128 (2nd Cir. 1995). Research

9 findings are often replicated in different populations and

10 consistency in these findings is an important factor in making a

11 judgment about causation. "Different studies that examine the

12 same exposure-disease relationship should yield similar results,"

13 whereas "[a]ny inconsistencies signal a need to question whether

14 the relationship is causal." "Reference Guide on Epidemiology"

15 at p. 162.

16   A "dose-response relationship" assumes the more intense the

17 exposure, the greater the risk of disease. Evidence of a dose-

18 response relationship strengthens the conclusion that the

19 relationship between the agent and the disease is causal, but it

20 is not necessary to infer causation. It is possible a dose-

21 response relationship may not be observed when there is a

22 threshold phenomenon. "Threshold phenomenon" means there is no

23 evidence of disease below a certain dose. Id. at p. 164.

24   "Biological plausibility" provides supporting evidence of

25 causation. Id. at p. 163. This factor asks whether it is

26 biologically plausible, in light of the biological and chemical

27 mechanisms involved, for exposure to the agent to precipitate the

28 **ORDER RE SUMMARY JUDGMENT-   17**

1   subsequent development of the disease. Asbestos Litigation, 52

2   F.3d at 1129. As noted above, "biological plausibility" is a

3   factor upon which plaintiffs' counsel place great reliance.

4   Indeed, in the context of radiation and certain cancers and

5   neoplastic diseases, this factor may well lend significant

6   support that radiation **can cause** the cancer or disease. However,

7   "biological plausibility" is not the same as "biological

8   certainty" that radiation, and specifically Hanford radiation,

9   caused cancer or disease in a specific individual. Such

10 certainty cannot be attained. "Biological plausibility" is but

11 one component of epidemiological proof.

12   An association exhibits "specificity" if the exposure is

13 associated only with a single disease or a single type of

14 disease. "Reference Guide on Epidemiology" at pp. 163-64.

15 "Specificity" is problematic with radiation exposure since such

16 exposure is associated with more than a single disease or a

17 single type of disease. However, although the presence of

18 specificity strengthens the inference of causation, its absence

19 does not weaken the inference. Id. at p. 163.

20   "Coherence" involves the analysis of the instant causal

21 factor in the context of other possible causal factors.

22 Alternative explanations and confounding factors should be ruled

23 out to avoid reaching an erroneous conclusion, although "it is

24 never possible to rule out every alternative explanation." Id.

25   If an exposure causes disease, the exposure must occur

26 before the disease develops. Obviously, if the exposure occurs

27 after the disease develops, it cannot cause the disease. Id. at

28 **ORDER RE SUMMARY JUDGMENT-   18**

1   p. 162.   This factor is known as "temporality."

2   Two absolute requirements for inferring an agent **can cause** a

3   disease is that exposure precede the disease and there be some

4   degree of statistical association between exposure and the

5   disease as manifested by "relative risk."   Thompson, "Causal

6   Inference in Epidemiology: Implications for Toxic Tort

7   Litigation," 71 **N.C. L. Rev.** 247 (1992) at p. 12.[11]

8   An **epidemiologist** does not need to find a relative risk of

9   2.0- a "doubling of the risk"[12]- for the purpose of concluding

10   an agent **can cause** a disease.   A "relative risk" of less than

11   2.0, along with other supporting evidence of causation such as

12   strong biological plausibility and the existence of a dose-

13   response relationship may be sufficient to infer that an agent

14   **can cause** a disease.   However, it is worth noting that

15   epidemiologists consider any relative risk ratio **below three**

16   **(3.0)** to indicate a weak causal association.   Any risk ratio

17   close to 1.0 is considered non-existent or extremely weak.[13]   A

18   relative risk ratio between 3.0 and 8.0 is considered "moderate,"

19   while anything above 8.0 is considered "strong."   Thompson 1992

20   at pp. 3-4.

21   _____

22   [11]   Hereinafter, "Thompson 1992."

23   [12]   "Doubling of risk" means the risk of the condition
     because of exposure is doubled over the background incidence of
24   the condition in the unexposed population.

25   [13]   A relative risk of 1.0 or less means the background
     incidence of disease in the unexposed population is equal to or
26   less than the incidence of disease from exposure to the agent in
     question.   It means there is not a causal association between
27   exposure and the disease.

28   **ORDER RE SUMMARY JUDGMENT-   19**

1   While it is necessary to establish that radiation **can cause**

2   the various cancers and diseases claimed by plaintiffs in this

3   case, that does not answer the ultimate question of whether

4   it can be considered a cause of the cancer or disease in a

5   particular individual. Of course, that is the very question

6   which this litigation seeks to answer:

7   The plaintiff must establish not only that
            the defendant's agent is capable of causing
8   disease but also that it did cause the plaintiff's
            disease. . . . **This question is not a question**
9   **about which an epidemiologist would have any**
            expertise to contribute. Rather it is a _legal_
10   **question . . . .**

11 "Reference Guide on Epidemiology" at p. 167 (Emphasis added).

12   "Doubling of the risk" is the **legal** standard for evaluating

13 the **sufficiency** of the plaintiffs' evidence and for determining

14 which claims should be heard by a jury. It does **not** however

15 establish disease causation at either the population or the

16 **individual** level. Thompson 1992 at p. 13. It is simply a means

17 which the Ninth Circuit has determined is proper for inferring

18 whether an agent is a "more likely than not" cause of a disease.

19 "Doubling of risk" reflects a policy judgment which seeks to be

20 fair to both plaintiffs and defendants. As the Ninth Circuit

21 explained in _Daubert II_:

22   No doubt, there will be unjust results under
            this substantive standard. If a drug increases
23   the likelihood of birth defects, but doesn't
            more than double it, some plaintiffs whose
24   injuries are attributable to the drug will be
            unable to recover. There is a converse un-
25   fairness under a regime that allows recovery
            to everyone that **may** have been affected by
26   the drug. Under this regime, all potential
            plaintiffs are entitled to recover, even though
27   most will not have suffered an injury that can

28 **ORDER RE SUMMARY JUDGMENT-   20**

1
    be attributed to the drug. One can conclude
    that this unfairness is inevitable when our
2
    tools for detecting causation are imperfect and
    we must rely on probabilities rather than more
3
    direct proof.

4 43 F.3d at 1320, n. 13 (Emphasis in text).[14]

5   The In re Hanford plaintiffs **at some point** need to cross the

6 "doubling of risk" threshold before their claims can be

7 considered by a jury. The question is whether it is appropriate

8 to require them to make that showing now on a generic basis, or

9 whether this determination should await the conclusion of Phase

10 III individual causation discovery and be made on an individual-

11 by-individual basis.

12   For certain of the conditions claimed by plaintiffs,

13 plaintiffs' experts have offered "doubling doses"[15] which are

14 derived from epidemiological studies of various populations. For

15 certain other conditions, plaintiffs' experts have opined about

16 risk co-efficients or risk estimates from which "doubling doses"

17 can be calculated. The defendants contend the court should use

18 these **generic** "doubling doses" as the floor for determining

19 whether **any** plaintiff has a claim triable before a jury. These

20 doubling doses are "generic" in the sense that they do not apply

21 to any specific individual. According to defendants, a plaintiff

22

23   [14] Plaintiffs cannot complain that the "doubling of risk"
standard works a greater burden upon them than the defendants.
Recall that in the epidemiological literature, 2.0 is actually
24 considered a "weak" association between an agent and a disease.
Thus, requiring plaintiffs' epidemiological proof to show a
25 relative risk greater than 2.0 in order for their claims to be
considered by a jury is not an unduly lofty standard.

26

27   [15] The dose at which the risk of contracting the disease is
doubled.

28 **ORDER RE SUMMARY JUDGMENT-**   **21**

1 subjected to a dose of Hanford radiation in **excess** of the generic

2 "doubling dose" (51% or more) meets the "more likely than not"

3 sufficiency standard (quantum of proof) standard and is entitled

4 to have his/her claim heard by a jury. Conversely, a plaintiff

5 subjected to the generic "doubling dose" or less (50% or less)

6 does not meet the "more likely than not" standard and his/her

7 claim should be dismissed on summary judgment.

8   The plaintiffs contend "doubling doses," if at all

9 appropriate, can only be calculated on an individual-by-

10 individual basis because said doses vary according to individual

11 factors such as smoking, diet, past medical treatment, familial

12 history, lifestyle and occupational and other exposures.

13 Accordingly, plaintiffs suggest that until all of the specific

14 individual information is gathered by defendants at Phase III of

15 this litigation, there is no way to calculate individual

16 "doubling doses" for the purpose of determining if an

17 individual's claim should be heard by a jury.

18   Defendants respond that these individual factors go to the

19 issue of alternative causes for the condition claimed, which at

20 the generic causation stage of the proceedings, are ignored in

21 favor of the plaintiffs. According to defendants, these factors

22 can only increase the doubling dose. Although defendants concede

23 individual doubling doses could vary, they contend it is possible

24 to compute what is essentially a generic minimum doubling dose

25 level. Once that level is exceeded, defendants say it will be

26 incumbent upon the individual to disprove that other factors,

27 such as a history of smoking, make it **less** likely that exposure

28 **ORDER RE SUMMARY JUDGMENT-   22**

1 to Hanford emissions was a cause in fact of his/her cancer- i.e.

2 disprove that it **increases** the doubling dose level necessary to

3 show that Hanford emissions were "more likely than not" a cause

4 in fact of his/her cancer.

5   What ultimately persuades this court that generic doubling

6 doses can be used for evaluating the **sufficiency** of plaintiffs'

7 claims at this generic causation stage is the fact plaintiffs'

8 own experts use such doses as a framework for analyzing

9 causation. Risk co-efficients and doubling doses are part of the

10 proof plaintiffs have offered to get beyond the generic causation

11 stage and beyond summary judgment.

12   A. James Ruttenber, Ph.D., M.D, is one of the plaintiffs'

13 experts. In his 1995 report, "Regarding Causal Relations Between

14 Exposure to Iodine-131 from Hanford and Thyroid Disease," at p.

15 6, he states:

16   One way to look at causation is to recognize
             that there is a background incidence for every
17   disease and that in order for an exposure to be
             implicated as the cause of a disease, it must
18   produce on its own, a risk equal to or greater
             than the background incidence for the disease.
19   Evidence for causation is thus produced
             by showing that, in one or more studies of
20   populations exposed to the agent of interest, there
             is a doubling of the disease rate over the rate of
21   a control or comparison group. Data from studies
             of exposed populations can be applied to an exposed
22   individual in order to determine his _a priori_ risk[16]
             for disease. For such an extrapolation to be valid,
23   the individual must have had an exposure that is

_____

24

       [16] An _a priori_ probability can be estimated for a dose or
25 range of doses for either a group of persons or a single
   individual. This type of probability or risk estimate is used to
26 make quantitative predictions and is the basis for comparing the
   risks of an exposed population or individual to the risk for an
27 unexposed population. (Ruttenber Report at p. 5).

28 **ORDER RE SUMMARY JUDGMENT-   23**

1   similar to the exposure for the populations from
           which the risk estimates were obtained, and have
2   similar susceptibilities to disease.

3   Dr. Radford provides "causative dose ranges" for thyroid

4 cancer. He assumes that an excess relative risk[17] of 100% is

5 sufficient to establish causality of thyroid cancer by radiation.

6 Radford considers this approach- a 100% increase in relative risk

7 or a "so-called doubling of the relative risk"- to be

8 "conservative." (Radford 1995 Iodine Report, "Comments On the

9 Medical Findings Associated With Exposure to Radioactivity From

10 the Hanford Facility in Washington," at p. 25). He offers this

11 same approach as a means for providing quantitative estimates of

12 the radiation dose required to increase the risk of non-thyroid

13 cancers by 100%. (Radford 1996 Non-Iodine Report, "Report on

14 Medical Effects From Radionuclides Other than Radioiodine

15 Discharged from the Hanford Nuclear Facility in Washington," at

16 p. 8).

17   As will be discussed in detail, Dr. Radford's risk co-

18 efficients and doubling doses are derived from "heterogeneous"

19 populations which include individuals with a range of different

20 susceptibilities to cancer. Radford does not believe the Hanford

21 population is so distinct from the populations studied

22 epidemiologically that the risk co-efficients and doubling doses

23 derived therefrom cannot be extrapolated to the Hanford

24 _____

25   [17] A relative risk of 1.0 means the rate of disease is the
   same in both the exposed and the unexposed populations. In that
26 case, the **excess relative risk** is zero. If the relative risk is
   2.0, the **excess relative risk** is 1.0. Excess relative risk
27 equals relative risk minus 1.0.

28 **ORDER RE SUMMARY JUDGMENT-   24**

1 population.   Nor is there any indication Dr. Ruttenber disagrees

2 with such in opining about the doubling doses for clinical and

3 subclinical hypothyroidism.

4   It is critical to point out that these "doubling doses" are

5 **not** and **cannot be** used to determine causation as to any **specific**

6 individual.[18] The court is simply employing them as a generic

7 threshold for determining whether **any** plaintiff has been exposed

8 to a dose of Hanford emissions sufficient to justify an **inference**

9 that those emissions were a probable cause of his/her disease,

10 not merely a possible cause.   This order does not dismiss any

11 specific individual plaintiffs.   It sets generic standards, based

12 on plaintiffs' own expert evidence, for determining which claims

13 should be heard by a jury.   Any plaintiff who meets the standard

14 must still prove to a jury that his/her exposure was a **cause in**

15 **fact** of his/her particular disease.   Such proof may take the form

16 of a differential diagnosis from a treating physician.

17   The court believes the use of "doubling doses" in this

18 manner represents an appropriate use of epidemiological evidence

19 within the law as set forth by the Ninth Circuit and the

20 Washington courts.[19] **Epidemiological** evidence in the form of

21 _____

22 [18] This is a recognition once again that epidemiology
   cannot answer questions about individual causation. However, the
   law must answer those questions and epidemiological proof is a
23 necessary component of the inquiry where actual cause cannot be
   determined via direct proof.

24

25 [19] Plaintiffs cite a number of cases from outside these
   jurisdictions in an effort to persuade the court a "doubling of
26 risk" standard is inappropriate for assessing the **sufficiency** of
   their proof at this **generic** stage of the proceedings. Two
27 examples are <u>Allen v. United States</u>, 588 F. Supp. 247 (D. Utah

28 **ORDER RE SUMMARY JUDGMENT-   25**

1 risk estimates and "doubling doses" cannot answer questions about

2 individual causation. However, those questions must ultimately

3 be resolved in some manner in a court of **law**. A necessary

4 **component** for resolving those questions is epidemiological proof

5 showing the dose level at which the risk of disease is doubled.

6 This is due to the undeniable fact that actual cause, what

7 actually occurred because of exposure to Hanford radiation

8 emissions, cannot be determined. Epidemiology is used to set a

9 reasonable benchmark for evaluating the **sufficiency of proof**. It

10 is not determinative of individual **causation** and this court does

11 not propose to use it in that manner. The court agrees with Dr.

12 Radford's statement that relative risk provides only a "point of

13 departure" for analyzing causation in individual cases. (Radford

14 Declaration, Ex. 5 to Plaintiffs' Appendix I re Non-Iodine

15 Claims, at pp. 2-3). The **sufficiency** of proof, as judged by the

16 "doubling of risk" standard, is distinct from the standard for

17 proving individual causation.

18   To this end, the use of "doubling doses" comports fully with

19 what this court envisioned would happen upon completion of the

20 expert record in Phase II. The fact plaintiffs' experts supply

21 such doses is confirmation plaintiffs understood the type of

22 screening this court anticipated. From the very outset of this

23 litigation, the court expressed to counsel that causation would

24 be the seminal issue in this litigation. The "doubling of risk"

25 _____

1984), and In re TMI Litigation Consolidated Proceedings, 927 F.

26 Supp. 834 (M.D. Pa. 1996). In those cases, the issue was
causation in fact at the individual causation stage of the

27 **proceedings**, a distinct inquiry discussed infra.

28 **ORDER RE SUMMARY JUDGMENT-   26**

1 standard is a practical and efficient way to assess the

2 sufficiency of proof gathered by counsel over the past number of

3 years. Assessing that proof solely under a "capable of causing"

4 standard would constitute a waste of effort and significantly

5 further delay the resolution of plaintiffs' claims. Again, this

6 is because proving radiation is "capable of causing" a disease

7 does not entitle any plaintiff to get his/her case before a jury.

8 He/she must still overcome the "doubling of risk" hurdle.

9   The time is **now** for determining which claims can proceed

10 before a jury. The next step in this litigation should be the

11 calculation of the doses received by the various plaintiffs.

12 From this, it can be determined who received a dose in excess of

13 the applicable "doubling dose" and therefore, have his/her claim

14 heard by a jury. Granted there may be a need for some additional

15 discovery pertaining to the particular individual (i.e. a

16 deposition of the individual and of his treating physician,

17 etc.), but this can be accomplished relatively quickly.

18

19   **B.   Causation In Fact Standard/Individual Causation Stage**

20   That plaintiffs may raise an inference radiation is "more

21 likely than not" a cause of their diseases does **not** mean they

22 have satisfied their burden of proving Hanford radiation

23 emissions are a **cause in fact** of their diseases. Although they

24 may satisfy their burden of producing sufficient evidence to

25 warrant a jury hearing their claims, they still bear the burden

26 of proving causation in fact to the satisfaction of a jury.

27   The plaintiffs apparently recognize this distinction, having

28 **ORDER RE SUMMARY JUDGMENT-**   **27**

1 asserted at various points in their briefs and at oral argument

2 that the causation standard is whether it is **more likely than not**

3 that Hanford emissions were a "substantial factor" in causing a

4 particular plaintiff's disease. "More likely than not"

5 represents the necessary factual quantum of proof (preponderance

6 of the evidence), whereas "substantial factor" is a term of

7 "legal significance." Keeton, et al., <u>Prosser and Keeton on</u>

8 <u>Torts</u>, (5th Ed. L. Ed. 1984) at p. 267. "The plaintiff must

9 introduce evidence which affords a reasonable basis for the

10 conclusion that it is **more likely than not** that the conduct of

11 the defendant was a **cause in fact** of the result." <u>Id</u>. at p. 269

12 (emphasis added).[20] There are two rules for proving "cause in

13 fact:" 1) "but for" and 2) "substantial factor."

14   Plaintiffs contend the "substantial factor" causation

15 standard should apply. They assert the Washington Supreme Court

16 would utilize a substantial factor standard in analyzing

17 **individual** causation in this case. Plaintiffs say a jury should

18 be able to award damages against the contractor defendants if it

19 finds Hanford emissions were a material element and a substantial

20 factor in causing their various diseases. According to

21 plaintiffs, they should not be required to prove to a jury that

22 their diseases would not have occurred "but for" Hanford

23

24

25
_____

26   [20] Unless it is a "more likely than not" cause, it cannot
be either a "but for" cause of the harm or a "substantial factor"

27 in causing the harm.

28 **ORDER RE SUMMARY JUDGMENT-   28**

1 emissions.[21] Defendants contend "but for" is the standard for

2 causation in fact.

3   Determining the causation in fact standard which a jury will

4 consider in assessing individual cases is not critical to the

5 inquiry currently before this court which concerns the quantum of

6 proof necessary to even get a case before a jury. In addition,

7 it is inappropriate to make a **general** ruling now on the causation

8 in fact standard to be applied in each and every **individual** case.

9

10   **C. Motion for Certification**

11   Plaintiffs have filed a motion asking this court to certify

12 to the Washington State Supreme Court the following question:

13   Whether, under Washington law, in a consolidated
            personal injury case arising from plaintiffs'
14   exposures to radiation over time in various
            geographic areas surrounding the Hanford Nuclear
15   Weapons Reservation, each plaintiff must
            establish causation by evidence that he or she
16   was exposed to a dose of radiation sufficient
            to be a substantial or significant factor in
17   causing the disease, or any part of the cause
            of the disease, or whether each plaintiff must
18   rather establish as a threshold matter, that he
            or she received a dose that would double the
19   risk of contracting the plaintiff's disease for
            all persons in a population exposed to that dose.
20
       Daubert II leaves no doubt that in a case which requires
21
   epidemiological evidence to prove causation, said evidence must
22
   show a "doubling of the risk" from exposure to the agent in
23
   question in order to infer the exposure is a "more likely than
24

25 _____

       [21] Prosser & Keeton on Torts at p. 266 describes the "but
26 for" test as a situation where the harm **would** not have occurred
   without the defendants' conduct or put another way, the harm
27 **would** have occurred even without the defendants' conduct.

28 **ORDER RE SUMMARY JUDGMENT-   29**

1 not" cause of injury. Otherwise, the evidence is insufficient to

2 warrant its consideration by a jury. The Ninth Circuit has

3 indicated this is also the law in the State of Washington:

> Under Washington tort law, a plaintiff must show
> that "the act complained of 'probably' or 'more
> likely than not' caused the subsequent disability."
> O'Donoghue v. Riggs, 73 Wash. 2d 814, 440 P.2d 823,
> 830 (1968). This is virtually the same as the
> standard under California tort law applied in
> Daubert II. See 43 F.3d at 1320. Under this
> standard, we held in Daubert II that expert
> testimony offered to prove causation did not
> satisfy the relevance requirement because the
> evidence suggested only that use of the drug at
> issue "could possibly have caused plaintiffs'
> injuries," rather than "more likely than not"
> caused the injuries, i.e., that use of the drug
> more than doubled the likelihood the injuries
> would occur. 43 F.3d at 1320-22.

Schudel, 120 F.3d at 996.

Because this court believes the Ninth Circuit has plainly

spoken about the evidentiary threshold applicable in the State of

Washington in cases involving epidemiological proof, it would be

inappropriate for **this court** to certify any question in that

regard to the Washington State Supreme Court. Plaintiffs'

request for certification should be directed, at the appropriate

time, to the Ninth Circuit Court of Appeals.

A separate and distinct question is whether the causation in

fact standard is one of "but for" or "substantial factor." We

have yet to reach the stage where cases are to be submitted for

jury consideration. The "doubling of risk" evidentiary threshold

must first be satisfied and an additional period of discovery is

contemplated with regard to the specific individuals who meet the

threshold requirement.

ORDER RE SUMMARY JUDGMENT–   30

1 Plaintiffs' Motion for Certification (Ct. Rec. 1125) is

2 **DENIED.**

3

4 **D. Summary**

5 In order to have their claims heard by a jury, plaintiffs

6 must produce evidence sufficient to raise an inference that their

7 exposure to Hanford emissions is a "more likely than not" cause

8 of their injuries. Because it can never be directly proven that

9 radiation was the cause of any plaintiff's injury,

10 epidemiological proof is necessary. As such, the "more likely

11 than not" standard can only be met by a showing the radiation

12 exposure "doubled the risk" of injury. **The admissibility and**

13 **sufficiency of plaintiffs' expert evidence will be assessed in**

14 **light of this standard.**

15

16 **V.   CROSS-RELIANCE ON EXPERTS BY PLAINTIFF GROUPS**

17 An issue has arisen as to whether the <u>Evenson</u> plaintiffs may

18 rely upon evidence from experts retained by the <u>Jaros</u>

19 plaintiffs[22] and conversely, whether the <u>Jaros</u> plaintiffs may

20 rely upon evidence from experts retained by the <u>Evenson</u>

21 plaintiffs.

22 Plaintiffs assert defendants cannot treat the <u>Jaros</u> and

23 <u>Evenson</u> groups independently for summary judgment purposes

24 because all of their claims have been consolidated for pretrial

25 purposes pursuant to Pretrial Order No. 1.   (Ct. Rec. 1).

26 _____

[22]   The <u>Jaros</u> group also includes the <u>Hamilton</u> and <u>Criswell</u>

27 plaintiffs.

28 **ORDER RE SUMMARY JUDGMENT-   31**

1   Furthermore, say plaintiffs, because of the common issues which

2   apply to all claimants, each claimant group can rely on the

3   submissions of each other (including evidentiary submissions),

4   and the admissions of defendants as to one set of plaintiffs can

5   be deemed admissions as to all plaintiffs.

6   Defendants contend Pretrial Order No. 1 is irrelevant,

7   relates solely to the filing of materials with the court, and

8   sets conditions for identifying materials pertaining to some or

9   all of the claims. Defendants note plaintiffs did not indicate

10   on their expert reports that they were being submitted on behalf

11   of plaintiff groups other than the plaintiff group or counsel

12   sponsoring the report. Defendants observe that even when cases

13   are consolidated pursuant to Fed. R. Civ. P. 42(a), they retain

14   their separate identity and each party is responsible for

15   complying with procedural requirements. Enterprise Bank v.

16   Seattle, 21 F.3d 233, 235 (8th Cir. 1994); Patton v. Aerojet

17   Ordnance Co., 765 F.2d 604, 606 (6th Cir. 1985). Finally,

18   defendants contend the plaintiffs are not simply sharing

19   resources pursuant to a carefully coordinated effort that had the

20   goal of efficiency and economy. Rather, defendants state the

21   Jaros and Evenson groups have taken contradictory positions on

22   the conditions at issue, the framework for analyzing causation,

23   the kind of evidence needed to prove causation, the amount of

24   iodine emitted from Hanford, the doses that resulted, and the

25   approach that should be used to estimate emissions and doses.

26   There are indeed some differences in the analyses and

27   conclusions of the Jaros and the Evenson experts with respect to

28   **ORDER RE SUMMARY JUDGMENT-**   **32**

1 certain topics (i.e. source term). Some of these differences are

2 pointed out _infra_ in the discussion regarding the plaintiffs'

3 expert evidence. It is the existence of these differences that

4 gives rise to the controversy whether _Jaros_ and _Evenson_ counsel,

5 until very recently, intended a coordinated approach to the

6 causation issue.

7   As a general rule, the court agrees that the mere fact

8 expert reports differ does not necessarily mean one of the

9 reports is scientifically unreliable. However, it is also

10 possible that such a difference manifests a methodological

11 unsoundness in one of the reports. Indeed, defense counsel have

12 noted such differences among the plaintiffs' various expert

13 reports in an attempt to discredit certain of the reports.

14 Defendants have had sufficient opportunity to identify these

15 differences in mounting their attack upon the plaintiffs' expert

16 reports.

17   The critical thing about the expert reports is whether they

18 present evidence which is scientifically reliable and relevant to

19 the inquiry before the court. _Daubert v. Merrell Dow_

20 _Pharmaceuticals, Inc._, 509 U.S. 579, 113 S.Ct. 2786 (1993). The

21 court has undertaken that analysis. As a result of that

22 analysis, defendants need not concern themselves with the

23 continued existence of any conflict between the evidence from the

24 experts retained by the _Evenson_ plaintiffs and from the experts

25 retained by the _Jaros_ plaintiffs. In sum, whether or not

26 conflicts continue to exist, the court discerns no prejudice to

27 defendants from the _Evenson_ plaintiffs relying on _Jaros_ experts

28 **ORDER RE SUMMARY JUDGMENT-   33**

1   and Jaros plaintiffs relying on Evenson experts for purposes of

2   these summary judgment motions.[23]

3   In addition, the claims of the Evenson plaintiffs and the

4   claims of the Jaros plaintiffs present a common issue of fact for

5   generic causation purposes: at what dose is the risk of a

6   certain disease for **any** plaintiff doubled as a result of Hanford

7   radiation exposure? This is a generic inquiry. Both the Jaros

8   evidence and the Evenson evidence is geared toward that inquiry.

9   Because the plaintiffs are similarly situated- all of them

10 allegedly exposed to Hanford radiation which they blame for their

11 diseases- they should be similarly treated for generic causation

12 summary judgment purposes. A segregation of the evidence for

13 strictly procedural reasons could potentially lead to results

14 which are substantively unjust. For example, a Jaros plaintiff

15 exposed to a dose of Hanford radiation exceeding the doubling

16 dose found in the admissible Jaros expert evidence gets to

17 proceed to trial. However, an Evenson plaintiff, **exposed to the**

18 **same dose of Hanford radiation**, does not get to proceed to trial

19 because the Evenson expert evidence either is admissible and

20 supports a higher doubling dose, or is inadmissible and therefore

21 establishes no doubling dose at all.

22   While the court is not entirely convinced Jaros and Evenson

23 _____

24   [23] As plaintiffs point out, they bear the risk of jury
    confusion arising from the presentation of conflicting expert
25 testimony at trial (testimony which has already passed Daubert
    scrutiny). Defendants would certainly have an opportunity to
26 "play" the experts off one another during cross-examination at
    trial in an attempt to impeach one or both of the experts before
27 a jury.

28 **ORDER RE SUMMARY JUDGMENT-**   **34**

1 counsel intended at the outset a coordinated approach to the

2 causation issue, the equities tip in favor of viewing their

3 expert evidence as a whole for summary judgment purposes.[24]

4

5 **VI.   PLAINTIFFS' EXPERT EVIDENCE**

6   Plaintiffs' expert evidence deals in general with the

7 following types of health effects: thyroid cancer (including

8 thyroid nodules and adenomas); various non-thyroid cancers; and

9 non-neoplastic thyroid disease including hypothyroidism

10 (including biochemical or subclinical variety), hyperthyroidism,

11 Graves' disease, goiter, and autoimmune thyroid disease

12 (autoimmune hypothyroidism, thyroiditis, etc.).

13   Plaintiffs attribute these health effects to either

14 radioiodine (I-131) exposure; exposure to radionuclides other

15 than I-131, chiefly plutonium; exposure to hexavalent

16 chromium[25]; or some combination thereof. Plaintiffs contend

17 they were exposed due to Hanford emissions to the air or to the

18 Columbia River, or some combination thereof.

19

20   **A.   Daubert Standard**

21   In Daubert v. Merrell Dow Pharmaceuticals, Inc., (Daubert

22 I), 113 S.Ct. 2786 (1993), the Supreme Court set forth the

23 standard for determining the admissibility of expert scientific

---

24   [24] The court is also mindful of the fact that the Jaros and

25 Evenson plaintiffs, although comprising the vast majority of the
   plaintiffs in this consolidated litigation, are not the only

26 plaintiffs.

27   [25] This is a toxin exposure, not a radiation-type exposure.

28 **ORDER RE SUMMARY JUDGMENT-   35**

1 evidence. <u>Daubert</u> entails a two part analysis. First, the court

2 must determine whether the expert's testimony reflects scientific

3 knowledge, whether his/her findings are derived by the scientific

4 method, and whether the work product amounts to good science.

5 This is also known as the "reliability" requirement. Secondly,

6 the court must ensure the proposed expert testimony logically

7 advances a material aspect of the proposing party's case. This

8 is known as the "fit" or "relevancy" requirement. <u>Daubert II</u>, 43

9 F.3d at 1315 citing <u>Daubert I</u>, 113 S.Ct. at 2795-97.

10   <u>Daubert's</u> two-part analysis is derived from FRE 702 which

11 says:

12   If scientific, technical, or other specialized
           knowledge will assist the trier of fact to
13   understand the evidence or to determine a fact
           in issue, a witness qualified as an expert by
14   knowledge, skill, experience, training, or
           education, may testify thereto in the form of
15   an opinion or otherwise.

16 If an individual is not qualified "by knowledge, skill,

17 experience, training or education" to render an opinion on a

18 particular question or subject, it follows that his/her opinion

19 cannot assist the trier of fact with regard to that particular

20 question or subject. <u>Whiting v. Boston Edison Co.</u>, 891 F.Supp.

21 12, 24 (D. Mass. 1995) ("Just as a lawyer is not by general

22 education and experience qualified to give an expert opinion on

23 every subject of the law, so too a scientist or medical doctor is

24 not presumed to have expert knowledge about every conceivable

25 scientific principle or disease").

26   In order to qualify as scientific knowledge, an inference or

27 assertion must be derived by the scientific method. "Scientific

28 **ORDER RE SUMMARY JUDGMENT-   36**

1   knowledge" does not require absolute certainty, but it does

2   require that an inference or assertion be derived by the

3   scientific method. The court's task is not to analyze what the

4   experts say, but what basis they have for saying it. Daubert II,

5   43 F.3d at 1316.

6   In other words, the court is to focus on the expert's

7   reasoning and not his conclusions. If the expert's reasoning

8   (methodology) is not sound, his conclusions are not admissible.

9   If the expert's conclusion is derived by sound scientific

10   methodology, the persuasiveness or "correctness" of the

11   conclusion is for the trier of fact to determine. The trier of

12   fact determines the "weight" to be afforded the conclusion. This

13   assumes of course the expert's conclusion is also "relevant" to

14   the inquiry at hand in that it logically advances a material

15   aspect of the proposing party's case. "Admissibility" and

16   "relevancy" are evidentiary matters for the court's

17   determination. "Weight" is a substantive matter falling within

18   the charge of the trier of fact.

19   In determining "admissibility," the court must satisfy

20   itself that the scientific evidence meets a certain standard of

21   reliability. The expert's bald assurance of scientific validity

22   is not enough. The party presenting the expert must show the

23   expert's findings are based on sound science, which requires

24   objective, independent validation of the expert's methodology.

25   Daubert II, 43 F.3d at 1316. The expert's testimony must be

26   based on "scientific knowledge," implying a "grounding in the

27   methods and procedures of science" and must connote "more than

28   **ORDER RE SUMMARY JUDGMENT-   37**

1   subjective belief or unsupported speculation." Hopkins v. Dow

2   Corning Corp., 33 F.3d 1116, 1124 (9th Cir. 1994), citing Daubert

3   I, 113 S.Ct. at 2795.

4   In Daubert II, the Ninth Circuit identified factors relevant

5   to the "reliability" determination. First is whether the expert

6   is proposing to testify about matters growing naturally and

7   directly out of research he has conducted independent of the

8   litigation, or whether his opinion has been developed for the

9   express purpose of offering testimony. If the testimony

10 proffered by an expert is based directly on legitimate

11 preexisting research unrelated to the litigation, this provides

12 the most persuasive basis for concluding the opinions expressed

13 were derived by the scientific method. Daubert II, 43 F.3d at

14 1317.

15   If the expert testimony is not based on independent

16 research, the party proffering it must come forward with other

17 objective, verifiable evidence that the testimony is based on

18 scientifically valid principles. One way of doing this is by

19 proof that the research and analysis supporting the proffered

20 conclusions have been subjected to normal scientific scrutiny

21 through peer review and publication. If the research is accepted

22 for publication in a reputable scientific journal after being

23 subjected to peer review, it is a "significant" indication it is

24 taken seriously by other scientists. Peer review and publication

25 increase the likelihood methodological flaws will be detected.

26 Id. at 1318 citing Daubert I, 113 S.Ct. at 2797.

27   According to the Ninth Circuit, the two principal ways for

28 **ORDER RE SUMMARY JUDGMENT-   38**

1 showing that evidence satisfies the first prong of the Daubert

2 analysis is if the proffered evidence grows out of pre-litigation

3 research or if the expert's research has been subjected to peer

4 review.   Where such evidence is unavailable, the proponent of

5 expert scientific testimony can attempt to satisfy its burden

6 through its own experts.   The experts must explain precisely how

7 they went about reaching their conclusions and refer to some

8 objective source such as a learned treatise, the policy statement

9 of a professional association, or a published article in a

10 reputable scientific journal.   This is necessary in order to show

11 they have followed the scientific method as practiced by a

12 recognized minority of scientists in the field.   Id. at 1318-19.

13 The expert's qualification, his conclusions, and his assurances

14 of reliability are not enough.   Id. at 1319.

15   Other admissibility factors enunciated by the Supreme Court

16 in Daubert I include whether the theory or technique employed by

17 the expert is generally accepted in the scientific community;

18 whether it can be and has been tested; and whether the known or

19 potential rate of error is acceptable.   Id. at 1316, citing

20 Daubert I, 113 S.Ct. at 2796-97.

21   "General acceptance" of a technique or theory hearkens back

22 to the discarded Frye test (Frye v. United States, 293 F. 1013

23 (D.C. Cir. 1923)):   is the methodology generally accepted in the

24 scientific community?   "General acceptance" is not the sine qua

25 non of admissibility under Daubert I.   Daubert focuses on the

26 reliability of the methodology.   Methods accepted by a minority

27 of the scientific community may well be sufficient.   Daubert II,

28 **ORDER RE SUMMARY JUDGMENT-   39**

1 43 F.3d at 1319, n. 11. Nonetheless, methodology which has only

2 attracted minimal support within the scientific community may

3 properly be viewed with skepticism. Daubert I, 113 S.Ct. 2797.

4   The Ninth Circuit opines that with regard to "derivative

5 analytical work," (experts who examine the available literature

6 and studies and draw conclusions from the original work), it

7 makes little sense to ask whether the technique can be and has

8 been tested, or what the known rate of potential error might be.

9 Id. at 1317, n. 4.

10   The second prong of the Daubert analysis is the "fit"

11 requirement or "relevancy" requirement. In order for expert

12 testimony to "fit" and be of assistance to the trier of fact, the

13 testimony must have a valid scientific connection to the

14 pertinent inquiry. Id. at 1320. The pertinent inquiry in this

15 consolidated litigation is whether radionuclides from Hanford are

16 a "more likely than not" cause of the health conditions claimed

17 by plaintiffs.[26]

18   To meet their burden of proving by a preponderance of the

19 evidence that their expert reports and the conclusions contained

20 therein are the product of sound scientific methodology,

21 Bourjaily v. U.S., 483 U.S. 171, 175-76 (1987), the plaintiffs

22 offer supporting affidavits from the experts who have prepared

23 the reports, as well as from additional experts who have not

24 prepared reports. In Daubert II, the Ninth Circuit observed that

25 it is appropriate for the proponent of scientific expert

26 _____

        [26] Obviously, "fit" is not a concern if the expert is not
27 qualified or has used unsound methodology.

28 ORDER RE SUMMARY JUDGMENT-   40

1 testimony to attempt to satisfy its burden through "the testimony

2 of its own experts."   43 F.3d at 1318-19.

3   This court is not aware of any prohibition against the

4 enlistment of additional experts who did not prepare expert

5 reports.   Plaintiffs assert the purpose of the affidavits is not

6 to alter or modify their existing expert reports and conclusions,

7 but to assist the court in its "gatekeeper" function and to

8 address the "flaws and misstatements" in defendants' <u>Daubert</u>

9 challenges.

10   The proffering of scientific testimony and making an initial

11 showing that it is derived by the scientific method enables a

12 party to establish a prima facie case of admissibility under FRE

13 702.   The opposing party is then entitled to challenge that

14 showing which it can do by presenting evidence, "including expert

15 testimony," that the proposing party's expert employed unsound

16 methodology or "failed to assiduously follow an otherwise sound

17 protocol."   <u>Id</u>. at 1318-19, n. 10.

18   Like the plaintiffs, the defendants have enlisted additional

19 experts who did not originally prepare reports on behalf of the

20 defendants.   Affidavits from these experts are included as part

21 of the defendants' replies to the plaintiffs' responses to the

22 motions in limine.[27]

23 _____

   [27]   FRE 104(a) provides that preliminary questions
24 concerning the qualification of a person to be a witness, the
   existence of a privilege, or the admissibility of evidence shall
25 be determined by the court and in making that determination, it
   is not bound by the rules of evidence.   The Advisory Committee
26 Notes to 104(a) indicate the court should hear **any relevant**
   evidence in making those determinations, including affidavits
27 which might otherwise be inadmissible at trial.

28 **ORDER RE SUMMARY JUDGMENT-   41**

According to the Ninth Circuit:

> Where the opposing party thus raises a
> material dispute as to the admissibility
> of expert scientific evidence, the district
> court must hold an in limine hearing
> (a so-called Daubert hearing) to consider
> the conflicting evidence and make findings
> about the soundness and reliability of the
> methodology employed by the scientific experts.

Id.   In this case, the parties agree an evidentiary hearing is unnecessary.   Accordingly, the court has proceeded to determine the motions in limine based on the voluminous written record (expert reports, expert depositions, etc.) before it.

B.   Radioiodine (I-131) Health Effects

1.   Lawrence Mayer

a.   Introduction

Dr. Mayer prepared a report in 1995 entitled "Biostatistical Issues in Connection with In re Hanford Nuclear Reservation Litigation."   Mayer refers to himself as a "clinically trained biostatistician."   He has degrees in medicine, statistics and biostatistics.   After completing medical school, he continued his graduate studies in statistics and completed his Ph.D in 1971. Thereafter, Mayer embarked on an academic career involving research in and teaching of statistics.

In 1979, he accepted a position at the University of Pennsylvania as Director of the Wharton Analysis Center (for the Evaluation of Energy Models), as Associate Professor of Statistics in the Wharton School, and with secondary appointments in Epidemiology in the School of Medicine, and in the School of

ORDER RE SUMMARY JUDGMENT-   42

1 Public and Urban Policy. Mayer states his work in the medical

2 school "focused on statistical analysis of data on problems in

3 endocrinology and metabolic activity." In 1982, he was a

4 Visiting Scholar at Stanford University where he "focused on

5 statistical computing and the methodology of assessing toxic

6 exposures."

7   Mayer is currently a Professor of Statistics in the

8 Department of Economics at Arizona State University (ASU); an

9 Adjunct Professor in the Schools of Public Health and Medicine at

10 Johns Hopkins University; and Chief Scientist in the Office of

11 Research at Good Samaritan Medical Center, Phoenix, Arizona.

12 Mayer says his research at ASU is in biostatistics and

13 epidemiology and he is "currently focusing on the use of

14 epidemiological measures such as attributable risk and prevented

15 fractions in longitudinal research designs for preventive

16 interventions." At Johns Hopkins, Mayer is a senior investigator

17 in the Prevention Research Center of the School of Hygiene and

18 Public Health. His research there "is on the biostatistical and

19 epidemiological methods used to analyze data from prospective

20 cohort studies of human development and disease processes." At

21 Good Samaritan, Mayer is "involved in research on the methodology

22 of preventive medicine and in teaching residents and fellows to

23 conduct research and to incorporate research results in clinical

24 practice."

25   Mayer was asked to give an expert opinion on the following

26 issues: 1) best estimate of the dose response relationship for

27 radiation and hypothyroidism; 2) the expected dose level of

28 **ORDER RE SUMMARY JUDGMENT-   43**

1 radiation for individuals that have a radiation related disease;

2 and 3) the issue of susceptibility and its incorporation into

3 radiation risk models. It is the first issue which is the focus

4 of defendants' motion in limine.

5   According to Mayer:

6   I have been asked to provide a rough estimate of
           the relationship between radiation dose and the
7   relative risk of hypothyroidism in a manner similar
           to that used in the HEDR modeling exercise. This
8   estimate relies on careful reading of the medical
           and epidemiological literature on the effects of
9   radiation on the thyroid, examination of the few
           epidemiological studies of the relationship between
10   radiation exposure and hypothyroidism, and my
           experience as a medical data analyst. **There is**
11   **not enough data to fit a formal statistical**
           analysis complete with prediction intervals,
12   **confidence intervals and hypothesis tests.**

13 (Mayer Rpt. at p. 5) (Emphasis added).

14   Mayer's analysis is based on data and information obtained

15 from articles regarding the Marshall Islanders, the survivors of

16 the Nagasaki bombing, and a series of papers on the effects of

17 therapeutic doses of radiation on the thyroid. From this data,

18 Mayer generated data points which were mapped onto a dose-re-

19 sponse curve ("a two parameter Weibull response curve"). Mayer

20 says that "[i]n many cases [the data points or values] were

21 **interpreted** from the article and not literally given by the

22 article." (_Id_. at p. 8)(Emphasis added).

23   Mayer's dose-response analysis does not distinguish between

24 biochemical and clinical cases of hypothyroidism, nor between

25 antibody positive and antibody negative cases of hypothyroidism.

26 He asserts the distinction can be left for "a later stage of the

27 proceedings, based upon specific information for individual

28 **ORDER RE SUMMARY JUDGMENT-   44**

1 plaintiffs."   Furthermore, because of the "sparsity of the data,"

2 Mayer states it is not possible "at this time" to account for

3 factors such as sample size, sampling scheme, gender distribu-

4 tion, type of radiation, and geographic differences.   (Id. at p.

5 6).

6   Based on his dose-response curve, Mayer concluded the dose

7 of **I-131 (radioiodine)** at which the risk of hypothyroidism

8 doubles for "a population" is approximately 50 rads.   Based on

9 his "uncertainty" analysis, he estimated the upper and lower

10 bounds of the doubling dose at between 30 to 80 rads.   Says

11 Mayer: "This type of upper and lower bounding is commonly used

12 [with] bioengineering problems **where there is not enough data to**

13 **permit an estimate of the variation in the data based on statis-**

14 **tical theory**" and "is an approximation comparable to the approxi-

15 mations found throughout the HEDR approach."   (Id. at p. 7)

16 (Emphasis added).

17

18   **b.   Reliability**

19   **(1) Condition At Issue: Autoimmune Hypothyroidism or Non-**

20   **Autoimmune Hypothyroidism**

21   Defendants criticize Mayer for his failure to clearly define

22 in his report what he means by "hypothyroidism," noting that he

23 does not distinguish between biochemical (subclinical) hypothy-

24 roidism, clinical hypothyroidism, and autoimmune thyroid dis-

25

26

27

28 **ORDER RE SUMMARY JUDGMENT-   45**

1 ease.[28]

2 Defendants observe that in his 1995 report, plaintiffs'

3 expert Dr. A. James Ruttenber distinguished between clinical and

4 biochemical hypothyroidism, and between autoimmune hypothyroidism

5 and non-autoimmune hypothyroidism. The etiology for non-autoim-

6 mune hypothyroidism involves direct cell-killing through I-131

7 exposure, whereas for autoimmune hypothyroidism the exposure

8 purportedly initiates an anti-thyroid autoimmune process leading

9 to cell damage and destruction (i.e. the body attacks its own

10 thyroid cells).

11 Plaintiffs contend there is no disagreement between Dr.

12 Mayer and Dr. Ruttenber because Mayer addresses hypothyroidism

13 induced by an **autoimmune process**, whereas Ruttenber addresses

14 hypothyroidism induced by direct cell-killing. Indeed, Dr.

15 Ruttenber's 1995 report[29] states as follows:

16 I conclude there is evidence for a doubling of
    the risk for biochemical hypothyroidism at
17 external radiation doses of 3.5 Gy (350 rad) and
    higher, and for clinical hypothyroidism at
18 external doses higher than 7.5 Gy (750 rad). **Since
    biochemical and clinical hypothyroidism as
19 defined above are not caused by neoplastic or
    autoimmune processes, it is possible that there
20 is a threshold below which disease would not be
    detected. For this reason, it is difficult to
21 comment on the risks for hypothyroidism below these
    dose levels.**

22

23

_____

24 [28] Hypothyroidism is a condition where the thyroid produces
    insufficient quantities of thyroid hormone, causing the body's
25 metabolic rate to slow down.

26 [29] "Report of A. James Ruttenber, Ph.D., M.D., Regarding
    Causal Association Between Exposure to Iodine-131 from Hanford
27 and Thyroid Disease."

28 **ORDER RE SUMMARY JUDGMENT-   46**

1 (Ruttenber 1995 Report at pp. 15-16) (Emphasis added).[30]

2   However, nowhere in Mayer's 1995 report is there any mention

3 of "autoimmune hypothyroidism" and indeed, Mayer states in his

4 report that he was not making any distinction regarding the types

5 of hypothyroidism. Nowhere have plaintiffs offered an explana-

6 tion for this omission. Furthermore, Mayer's report does not

7 contain any discussion of disease mechanisms and he testified

8 during his deposition that he was not an expert in such mecha-

9 nisms, including autoimmune mechanisms. (Mayer Dep. at 164).

10

11   **(2) Underlying Epidemiological Data**

12   Defendants contend Mayer's conclusions are based on

13 scientifically unreliable inferences drawn from epidemiological

14 data. Mayer used eleven different epidemiological studies as

15 sources for his data points (Mayer Rpt. at p. 8), however the

16 four critical studies are those involving low doses of radiation

17 (Kaplan, Nagataki, Maxon and Larsen). It is the low dose studies

18 from which Mayer derives his 50 rad doubling dose, with upper and

19 lower bounds of 30 to 80 rads. The other studies involved

20 exposure to doses in excess of 2,000 rads.

21 //

22 //

23

24

25

26   [30] In his report, Ruttenber also discusses chronic thyroid-
itis which he says may result in biochemical or clinical hypothy-
roidism and is most commonly caused by an autoimmune process.

27 Thyroiditis is a condition distinct from hypothyroidism.

28 **ORDER RE SUMMARY JUDGMENT-   47**

(a) Kaplan 1988[31]

Kaplan was a pilot study, the purpose of which was to evaluate the presence of thyroid conditions among 91 women treated with X-rays for tuberculosis. Kaplan found only one case of hypothyroidism among the 91 women for a prevalence of 1.38%. It found only one case in the unexposed control group consisting of 72 individuals (1.1% prevalence).

Rather than focusing on this hypothyroidism data, Mayer opted instead to focus on Kaplan's "broadly defined category" of "auto-immune thyroid disease." (Table 5 of Kaplan at p. 379). Therefore, say defendants, Mayer "misrepresented" the data.

In his deposition, Mayer offered this rationale for his use of the "auto-immune thyroid disease" category:

> [I]n this particular article [Kaplan], I used the
> auto-immune thyroid disease, which was as
> close as I could get, to give me a relative
> risk, and I assumed that for all hypothyroidism,
> that the ratio would be approximately the same.
> So I took the number from the auto-immune
> thyroid disease, not from . . . the clinical
> thyroid disease [clinical hypothyroidism].

(Mayer Dep. at pp. 161-62). According to Mayer, Kaplan's "hypo-thyroidism" category included only "explicit" or "clinical" hypothyroidism. (Mayer Dep. at pp. 162-63).

In his declaration, submitted in response to defendants' motion in limine (Plaintiffs' Ex. 5 to Appendix 1 re Iodine Claims), Mayer says the reason he ignored the category labelled

---

[31] Kaplan, et al., "Thyroid, Parathyroid, and Salivary Gland Evaluations in Patients Exposed to Multiple Fluoroscopic Examinations During Tuberculosis Therapy: A Pilot Study," 66 **Journal of Clinical Endocrinology and Metabolism** 376 (1988). (Defendants' Ex. 59).

ORDER RE SUMMARY JUDGMENT-   48

1   "Hypothyroidism" in Table 5 of Kaplan was because it could only

2   be referring to non-autoimmune hypothyroidism, whereas Kaplan's

3   "autoimmune thyroid disease" category would include certain types

4   of hypothyroidism, including antibody-positive hypothyroidism.

5   (Mayer Declaration at Paragraph 44).

6   Here again, it must be pointed out that there is nothing in

7   Mayer's report which says he was focusing on "autoimmune hypo-

8   thyroidism." There is not a single reference in the report to

9   autoimmune hypothyroidism. Furthermore, at his deposition, Mayer

10   was unable to explain the conditions falling within Kaplan's

11   category of "autoimmune thyroid disease." He acknowledged that

12   he was not an expert in this area. He also acknowledged that

13   this category would include diseases **other** than hypothyroidism.

14   (Mayer Dep. at pp. 164-65).

15   According to Dr. John D. Boice, Jr., the lead epidemiologist

16   on the Kaplan study, the "autoimmune thyroid disease" category

17   was "broad," including conditions having different etiologies.

18   States Dr. Boice:

19   This broad classification was not ideal.
             It included conditions with differing
20   etiologies (e.g. Hashimoto's thyroiditis
             and Graves' disease). We used it, however,
21   because the study was exploratory in nature
             and the kind of grouping could have laid
22   the groundwork for an expanded study that
             might have permitted a more focused analysis
23   on specific conditions.

24   (Boice Affidavit at Paragraph 26).[32] Boice indicates that "an

25   important aspect of epidemiological studies is to define the

26   _____

27   [32]   Defendants' Ex. 195.

28   **ORDER RE SUMMARY JUDGMENT-   49**

1   disease of interest as specifically as possible." (Id. at

2   Paragraph 24).[33]

3   In his declaration, Mayer acknowledges the Kaplan study's

4   "autoimmune thyroid disease" category includes diseases other

5   than hypothyroidism, but he [Mayer] says he preferred to use the

6   number Kaplan gave for that category (prevalence ratio of 2.2)

7   because it "avoids some of the problems with interview data and

8   allows for further **progression** to hypothyroidism." Mayer states

9   he used 2.2 as a "measure of relative risk for long-term develop-

10   ment of hypothyroidism." (Mayer Declaration at Paragraph 44).

11   Defendants correctly note that there is no mention of a "progres-

12   sion" theory in Mayer's expert report.

13   Michael Kaplan, M.D., an endocrinologist and the lead

14   clinician and author of the Kaplan study, asserts it is inappro-

15   priate to use the odds ratio of 2.2 as the estimate for the

16   relative risk of **autoimmune hypothyroidism**. Kaplan disagrees

17   with Mayer's theory that even if this figure overstates the risk

18   of autoimmune hypothyroidism observed in the study, some of the

19   conditions included in the "autoimmune thyroid disease category"

20   would progress to hypothyroidism over time. According to Kaplan:

21   The data generated in the study reflects
                    conditions that were actually observed at
22   a specific point in time. To make quantitative
                    predictions about what might happen to this
23   population in the future is unscientific. The
                    generalization that some persons in the exposed
24   group might have conditions that could evolve
                    into hypothyroidism provides no information

25   _____

26   [33] Boice does not deny, however, that autoimmune hypothy-
     roidism would be included in the "autoimmune thyroid disease"
27   category.

28   **ORDER RE SUMMARY JUDGMENT-   50**

> about how many of these might progress to this
> state or how the progression, if any, compares
> with the control population. This information
> can be obtained only through actual observation
> and study.

(Kaplan Declaration at Paragraph 24).[34]

Geoffrey R. Howe, Ph.D., one of the defendants' experts and the Professor and Head of the Division of Epidemiology in the School of Public Health at Columbia University, agrees:

> Dr. Mayer's argument that he can include data
> for a different condition than the one of interest
> on the assumption that those conditions will evolve
> over time to more serious conditions or into the
> condition of interest [autoimmune hypothyroidism]
> is justified <u>only</u> when it is known that a certain
> percentage of the less serious disease condition
> progress to the disease condition of interest and
> when it is known that such progression rates are
> independent of radiation exposure. Otherwise one
> cannot predict future risk of the more serious
> condition based on current risk of the less serious
> condition. In addition, the assumption that any
> radiation related relative risk will increase in the
> future as more cases are diagnosed amongst exposed
> individuals is invalid, since this ignores the fact
> that the assumed unexposed population will undergo
> some change as well, (i.e. additional cases will also
> develop in this unexposed group). The only valid way
> to compare the risk of the unexposed and exposed
> populations is to consider data from both populations
> at the same point in time. The fact that some
> conditions might progress into another condition does
> not provide any basis for adjusting data reported in
> the study of interest or for reaching conclusions about
> future prevalence and risk.

(Howe Affidavit at Paragraph 9).[35] Dr. Boice adds that "there is no inevitable progression to hypothyroidism and regression to the normal state is not uncommon." (Boice Affidavit at Paragraph 42).

---

[34] Defendants' Ex. 200.

[35] Defendants' Ex. 208.

ORDER RE SUMMARY JUDGMENT-   51

1   Defendants contend Mayer's reliance on the Kaplan study is

2 improper for several additional reasons. First, Kaplan's "auto-

3 immune thyroid disease" category is not statistically signifi-

4 cant. Although Kaplan reported a "prevalence ratio"[36] of 2.2

5 for this category, his 95% confidence interval was 0.8 to 6.2.[37]

6 (Kaplan 1988 at p. 379). This is not statistically significant

7 because the relative risk range includes 1.0- the background rate

8 (i.e. disease as likely to be produced by background factors as

9 by the agent in question). "Reference Guide on Epidemiology" at

10 p. 173 (definition of "Confidence Interval"). Secondly, say

11 defendants, Kaplan does not analyze dose-response relationships

12 and therefore, cannot be used to analyze such relationships.

13 Thirdly, defendants say while Mayer assumed an average dose in

14 the Kaplan study of 60 rads, Kaplan did not provide such a figure

15 and Mayer, in his report, does not explain how he arrived at that

16 figure. Furthermore, say defendants, reliance on Kaplan is

17 improper because that study did not account for potentially

18 significant doses that participants received from other sources,

19 including thyroid doses of up to several hundred rads.

20   Plaintiffs contend defendants neglect to mention that the

21 data point Mayer relied upon from Kaplan is statistically signif-

22 icant at the 90% confidence level. Mayer observes that Kaplan

23 gave a "p-value" of 0.096 for the prevalence ratio of 2.2 as-

24 _____

25   [36] "Prevalence ratio" is synonymous with "relative risk."

26   [37] This confidence interval indicates the range of relative
risk values that would result 95% of the time if the study were
27 repeated. "Reference Guide on Epidemiology," at p. 173.

28 **ORDER RE SUMMARY JUDGMENT-**   **52**

1 signed to the "autoimmune thyroid disease" category. Mayer

2 states this "p-value" means the probability of the increase

3 occurring by chance was less than 10% or, in other words, that

4 the increase was significant at the 90% confidence level.[38]

5   Mayer downplays the importance of statistical significance:

> Although defendants challenge the Kaplan study
> as not being statistically significant at the
> 95% confidence level, radiation is associated
> (via disease progression) with both biochemical
> and overt hypothyroidism at doses as low as 11 to
> 112 rads with a statistical significance exceeding
> the 90% confidence interval. Ninety-five percent
> statistical significance is not a sine qua non
> for association, especially when the biological
> basis for the association is clear-- as in this
> case --via the autoimmune route. It is important
> to note that the absence of significance does not
> translate into support for the null hypothesis
> of no association. A lack of statistical significance
> does not alter the best estimate of risk. **Whether
> 95% or 90% significant or not, Kaplan's estimate of
> relative risk of autoimmune thyroiditis is 2.2 at
> exposures to low-level radiation. This estimate
> is the best unbiased estimate of the risk regardless
> of its level of significance.**

(Mayer Declaration at Paragraph 47) (Emphasis added).

Defendants respond with the affidavit of Dr. Boice who says

the "autoimmune thyroid disease" category data was not even

---

[38] "P-value" (also known as "probability value") is the
probability of getting a value of the test statistic equal to or
more extreme than the result observed, given that the null
hypothesis is true. The letter "p," followed by the abbreviation
"n.s." (not significant) or by the symbol for less than (<) and a
decimal notation is a statement of the probability that the
difference observed could have occurred by chance. **In most
biomedical and epidemiological work, a study result whose proba-
bility value is less than 5% (p < 0.5) or less than 1% (p < 0.1)
is considered sufficiently unlikely to have occurred by chance to
justify the designation "statistically significant."** "Reference
Guide on Epidemiology" at p. 175-76. (Emphasis added).
The "p-value" in the Kaplan study for autoimmune thyroid
disease is 0.096 which is less than both 0.5 and 0.1.

ORDER RE SUMMARY JUDGMENT-   53

significant at a 90% confidence interval (with binomial methods,

the p-value was 0.076 with an odds ratio of 0.90-7.35; with the

Poisson test, the p-value was 0.089 with a relative risk range of

0.87-6.41).   (Boice Affidavit at paragraphs 36 and 37).   Defen-

dants further note that 95% confidence intervals are the norm in

epidemiological studies.   Indeed, Dr. Boice states that a 95%

confidence interval was used in the Kaplan study because autoim-

mune thyroid disease and other thyroid diseases have not been

clearly linked to radiation.   (Boice Affidavit at Paragraph 34).

        Mayer's assertion that statistical significance is not

important because the biological basis for association is clear

via the autoimmune route is curious in light of his deposition

testimony that it was only his "suspicion" that low doses of

radiation produce autoimmune disease leading to at least subclin-

ical hypothyroidism.   (Mayer Dep. at pp. 175-76).[39]

        In his declaration, Mayer states he used the odds ratio for

the "autoimmune thyroid disease" category as an estimate of the

relative risk of hypothyroidism because it was a "conservative

estimate" of the relative risk of hypothyroidism, and because the

relative response to radiation exposure for other autoimmune

diseases would be no greater than that for autoimmune hypothy-

roidism.   He adds that "[s]ince the defendants appear not to like

this analysis, I computed a doubling dose with the Kaplan data

point removed and found that the results are essentially un-

changed."   According to Mayer, removal of the Kaplan data had

---

        [39]   See discussion _infra_ regarding "biological plausibili-
ty."

ORDER RE SUMMARY JUDGMENT-   54

1 | virtually no effect on the doubling dose estimate from the

2 | Weibull model and that "this was no surprise, since the Kaplan

3 | relative risk is basically identical to the Nagataki relative

4 | risk." (Mayer Declaration at Paragraph 48).

5 | Rather than defending Mayer's use of the Kaplan data to the

6 | very end, the plaintiffs contend defendants' objections are moot

7 | because Mayer refit the model without the Kaplan data and found

8 | the results were unchanged. The fact Mayer is willing to change

9 | his analysis simply because defendants do not like his use of the

10 | Kaplan study does not inspire confidence in his methodology and

11 | indeed, suggests a focus on results rather than methodology.

12 | Drs. Boice and Kaplan are very candid and persuasive in

13 | pointing out the limitations of their **own** study- i.e. low partic-

14 | ipation rate of eligible subjects; unbalanced racial distribu-

15 | tions of the exposed and control groups which might be responsi-

16 | ble for differences detected in thyroid disorders, unrelated to

17 | the radiation received; exposed group had more serious and more

18 | advanced tuberculosis, had received more surgical treatments, and

19 | were older at examination, all of which could be responsible for

20 | differences detected in thyroid disorders, unrelated to the

21 | radiation received; uncertainties about the actual doses received

22 | by the study subjects; study included subjects who received

23 | radiation doses of up to 200 rads from other sources of exposure,

24 | such as thyroid scintiscan. (Boice Affidavit at Paragraphs 14,

25 | 17-23, 19, 21 and 22). Boice and Kaplan agree it is inappropri-

26 | ate and unscientific to rely on their data to infer that radia-

27 | tion doses of up to 112 rads cause autoimmune thyroid disease or

28 | **ORDER RE SUMMARY JUDGMENT-   55**

1 any of the specific conditions included in that category.

2 (Kaplan Declaration at Paragraph 16; Boice Affidavit at Paragraph

3 40).[40]

4   With regard to the failure of the Kaplan study to take into

5 account that some of the subjects received radiation doses of up

6 to 200 rads from other sources of exposure, such as thyroid

7 scintiscan, Mayer took this added dosage into consideration when

8 preparing his post-report declaration and calculated an addition-

9 al average dose to all of the exposed cases equal to 29 rads. He

10 concluded that even with the addition, the resulting doubling

11 dose did not move outside his range of error.   (Mayer Declaration

12 at Paragraphs 50-57).

13   Obviously, Mayer failed to detect this problem before

14 putting together the dose-response curve found in his report.

15 Epidemiologists Boice and Edward Radford, one of the plaintiffs'

16 experts, agree that ideally, subjects who had exposures from

17 other sources ("high dose subjects") should have been excluded

18 from the study.   (Boice Affidavit at Paragraph 22); (Radford Dep.

19 at pp. 427-28).

20 //

21 //

22

23

24 _____

   [40] Mayer was asked at his deposition whether he agreed the
25 Kaplan study had not concluded there was an association between
   low level radiation exposure and clinically significant autoim-
26 mune disease. (Kaplan at p. 381). His response was: "I don't
   know what Kaplan concluded. I only know what Kaplan wrote."
27 (Mayer Dep. at p. 183).

28 **ORDER RE SUMMARY JUDGMENT-   56**

(b) Nagataki 1994[41]

Nagataki analyzed thyroid abnormalities among atomic bomb
survivors in Nagasaki. In his report, Mayer cited Nagataki for
the proposition that at an "approximate dose" of 40 rads the
"approximate prevalence" of hypothyroidism is .02 (2.0 percent)
and at an "approximate dose" of 70 rads the "approximate
prevalence" is .025 (2.5 percent). (Mayer Rpt. at p. 8). Mayer
derived these figures from a curve depicted in Figure 2 of
Nagataki at p. 368 labelled "Antibody-Positive Spontaneous
Hypothyroidism."

According to defendants, although Nagataki deals with
several different conditions, including several different hypo-
thyroid conditions, Mayer focused only on the one autoimmune
condition called "antibody **positive** spontaneous hypothyroidism."
Defendants note that Mayer ignored the data pertaining to "anti-
body **negative** spontaneous hypothyroidism." Defendants point out
this is inconsistent with the statement in Mayer's report that
his analysis did not distinguish between "biochemical cases and
clinical cases **or between antibody positive and antibody negative
cases.**" (Mayer Report. at 6) (Emphasis added). During his
deposition, Mayer confirmed this statement made in his report.
(Mayer Dep. at p. 303).

Nagataki did not specifically report the dose and prevalence
data which Mayer includes in his analysis (.02 prevalence at 40
rads and .025 prevalence at 70 rads). Rather, Mayer looked at

---

[41] Nagataki, et al., "Thyroid Diseases Among Atomic Bomb
Survivors," 272 **The Journal of the AMA** 364 (Aug. 3, 1994).

ORDER RE SUMMARY JUDGMENT-   57

1 Nagataki's "Antibody-Positive Spontaneous Hypothyroidism" curve

2 and treated the odds ratio as synonymous with prevalence. On the

3 Nagataki curve, dose is measured by sieverts (Sv). One sievert

4 equals one hundred rads. Thus, where an odds ratio of 2.0

5 intersects with the "Antibody-Positive Spontaneous

6 Hypothyroidism" curve, Mayer estimated that to be about 40 rads.

7 Where the odds ratio of 2.5 intersects with the same curve, Mayer

8 estimated that to be about 70 rads.[42] Mayer referred to this as

9 a "rough interpolation." (Mayer Dep. at p. 230).

10   Defendants argue Nagataki's curve is "concave" and this is a

11 significant fact which Mayer ignored. Nagataki's "Antibody-

12 Positive Spontaneous Hypothyroidism" curve reaches a peak and

13 then starts decreasing as the dosage increases. Nagataki took

14 note of this, stating:

15   The present study has shown for the first
            time an increase in prevalence of autoimmune
16   hypothyroidism among atomic bomb survivors.
            The dose-response curve is **concave**, reaching
17   a maximum of 0.7 Sv [70 rads], **and thus
            indicates the necessity for further studies
18   on relatively low-dose radiation effects on
            thyroid disease.**

19
    (Nagataki at p. 370) (Emphasis added).
20
        Defendants state Nagataki's curve is the opposite of a
21
    _____

22   [42] Mayer treats odds ratio the same as prevalence. The
    odds ratio is similar to the relative risk ratio. "Reference
23 Guide on Epidemiology" at p. 149. The Reference Guide indicates
    that for all practical purposes, the odds ratio is comparable to
24 relative risk when the disease is rare. However, as the disease
    becomes more common, they diverge. Id.
25   It appears Mayer treats an odds ratio of 2.0 the same as a
    relative risk of 2.0 which is a doubling of the risk due to
26 exposure. This is how he arrives at the ultimate conclusion in
    his report that the doubling of the risk for hypothyroidism is
27 approximately 50 rads, with a range between 30 and 80 rads.

28 **ORDER RE SUMMARY JUDGMENT-   58**

1   "dose-response relationship" which assumes the more intense the

2   exposure, the greater the risk of disease.   Evidence of a dose-

3   response relationship strengthens the conclusion that the rela-

4   tionship between the agent and the disease is causal.   "Reference

5   Guide on Epidemiology" at p. 164.   Defendants contend Mayer

6   simply ignored the absence of a dose-response relationship.

7   Finally, defendants note that Mayer opined the most that

8   could be derived out of Nagataki was that "[i]t lends evidence to

9   an association" between radiation exposure and autoimmune hypo-

10 thyroidism.   Mayer conceded that not enough studies have been

11 completed in order to reach a "definitive conclusion."   (Mayer

12 Dep. at p. 275).   According to the "Reference Guide on Epidemiol-

13 ogy," exposure to an agent and disease are "associated" when they

14 occur more frequently together than one would expect by chance:

15 "Association implies a range of possible relationships, but it

16 does not necessarily imply a cause-effect relationship between

17 exposure and disease."   Id. at 147.   Mayer acknowledged that

18 "long before causality would be association."   (Mayer Dep. at p.

19 50).

20   Defendants contend the Nagataki study does not satisfy any

21 of the epidemiological criteria.   The first reason is because

22 the risk does not increase with dose.   Secondly, no other study

23 has duplicated the Nagataki results and therefore, the "consis-

24 tency" criterion is not met.   Indeed, according to Nagataki, his

25 study "has shown for the **first time** an increase in prevalence of

26 autoimmune hypothyroidism among atomic bomb survivors."   (Nagat-

27 aki 1994 at p. 370) (Emphasis added).   Thirdly, defendants claim

28 **ORDER RE SUMMARY JUDGMENT-   59**

1   the strength of the association is "weak" because the relative

2   risk is less than three. Mayer opines that the odds ratio

3   (similar to relative risk) peaks at 2.5 at a dose of 70 rads.

4   Defendants note that as an **epidemiological matter**, an association

5   below 3 is considered "weak." Finally, defendants contend the

6   biological mechanism by which radiation induces an autoimmune

7   response is unproven and remains only a theory. Hence, they say

8   there is not strong "biologic plausibility."

9   The plaintiffs contend Nagataki's curve is actually "con-

10 vex." However, Nagataki himself refers to his "antibody-positive

11 spontaneous hypothyroidism" curve as being "concave." Secondly,

12 regardless of whether it is "concave" or "convex," the fact is

13 that it reaches a peak and then declines.

14   Plaintiffs observe that the Nagataki curve qualifies as a

15 "dose-response curve" in accord with the definition of "Dose-

16 Response Relationship" contained in the "Reference Guide on

17 Epidemiology," at p. 174: "A relationship in which a change in

18 amount, intensity, or duration of exposure is associated with a

19 change- either an increase or a **decrease**- in risk of disease."

20 (Emphasis added). However, this ignores the fact that in terms

21 of epidemiological criteria for inferring causation, a dose-

22 response relationship is significant only **if the more intense the**

23 **exposure, the greater the risk of disease.** Id. at p. 164.

24 Evidence of such a dose-response relationship (more intense the

25 exposure, the greater the risk) strengthens the conclusion that

26 the relationship between the agent and the disease is causal,

27 although a dose-response relationship is not necessary to infer

28 **ORDER RE SUMMARY JUDGMENT-   60**

1 causation. Id.[43]

2   In his declaration, Mayer asserts that a dose-response curve

3 which partly increases and partly decreases is associated with

4 exposures that have multiple disease producing mechanisms.

5 (Mayer Declaration at Paragraph 37, n. 29). Plaintiffs say the

6 combination of early-onset hypothyroidism (via radiation-mediated

7 cellular and tissue damage) and late-onset hypothyroidism (via

8 autoimmune thyroid disease) is a perfect example of multiple

9 disease producing mechanisms.[44]

10   According to Mayer:

11   The convexity of Nagataki's response curve
     could be a function of statistical variability

12   or could be an accurate reflection of the
     dose response relationship. The former is

13   quite likely since there are few observations
     where the response declines. At this level,

14   there are only two cases for clinical hypo-
     thyroidism. I ignored the downturn in my

15   fit because the small number of cases at the
     high dose gives the associated response too

16   much variance to be considered reliable. But,
     there is a distinct possibility that the dose

17   response is convex. Although present to a
     certain extent in the Chernobyl antibody data,

18   I felt this was too speculative to justify a
     more complex dose response curve. The hypothesis

19   that the curve is convex because the effect of
     external radiation on the autoimmune reaction

20   of the body decreases at high dose is not

21 _____

   [43] Mayer's curve is of the "no-threshold" variety (exposure

22 can cause disease down to the very lowest doses) and therefore,
   the lack of a dose-response relationship (the more intense the

23 exposure, the greater the risk of disease) cannot be excused due
   to the existence of a threshold phenomenon (low dose exposure

24 does not cause disease until the exposure exceeds a certain
   dose). "Reference Guide on Epidemiology" at p. 164. See also

25 Mayer Dep. at pp. 320-21.

26 [44] The court notes, however, that the Nagataki curve deals
   only with "antibody-positive spontaneous hypothyroidism" which is

27 an autoimmune condition.

28 **ORDER RE SUMMARY JUDGMENT-**   **61**

1   unreasonable since at the very high doses the
           thyroid is totally destroyed and there may be
2   almost no autoimmune response.

3 (Mayer Declaration at Paragraph 37).

4   The defendants contend Mayer's assertion that there are only

5 two cases of clinical hypothyroidism at the area of the curve

6 where the response declines is false and misleading. They note

7 that Table 2 of Nagataki contains information for three dose

8 categories: less than 1 rad, 1 to 99 rads; and 100 or more rads-

9 and that the downturn in Nagataki's curve begins at 70 rads.

10 Consequently, defendants correctly point out that Mayer could not

11 know for sure how many cases of clinical hypothyroidism are

12 located at the downturn in the curve. This is because Nagataki

13 does not have a 70 to 99 rads category and it is possible the

14 bulk of the cases reported for the 1 to 99 rads category could be

15 located at a level of 70 rads and above. In the 1 to 99 rads

16 category for antibody positive spontaneous hypothyroidism,

17 Nagataki reports 25 cases, 11 which are clinical and 14 of which

18 are subclinical. Defendants have pointed out an errant

19 assumption on the part of Mayer which invalidates his reason for

20 "ignor[ing] the downturn in [his] fit."

21   Because in his report Mayer did not distinguish between

22 biochemical and clinical cases, defendants convincingly contend

23 he should not have ignored the subclinical (biochemical) cases

24 reported by Nagataki for antibody positive spontaneous

25 hypothyroidism at the 1-99 rads range and the 100 or more rads

26

27

28 **ORDER RE SUMMARY JUDGMENT-**   **62**

1 range.[45]

2

3   **(c) Larsen 1978[46]**

4   Larsen reported on thyroid conditions among a population in

5 the Marshall Islands that was exposed to radiation fallout from a

6 thermonuclear bomb test in 1954. In his report, Mayer cited

7 Larsen for the proposition that at an "approximate dose" of 400

8 rads, the "approximate prevalence" of hypothyroidism is .09 (9

9 percent). (Mayer Report at p. 8).

10   Defendants say Mayer does not explain in his report how he

11 derived this estimate from Larsen. Defendants assert Mayer

12 ignored Larsen's data for clinical hypothyroidism and based his

13 conclusion on sensitive biochemical measurements Larsen reported

14 for a single island, Rongelap.

15   According to Larsen at p. 102:

16   Despite the high prevalence of thyroid
            nodularity in Marshallese inadvertently
17   exposed to fall-out in 1954, only two
            subjects, both about one year of age at
18   exposure, have been found to have **primary**
            hypothyroidism.[47] The recent availability
19   of sophisticated immunoassay techniques for
            thyroxine (T4) and thyrotropin (TSH) has
20   allowed more thorough thyroid evaluation of
            the exposed population who do not have **known**
21   thyroid abnormalities **(43 Rongelap people).**

22 ────────────────

23   [45] At 100 rads and beyond, Nagataki reports seven cases,
    two of which are clinical and five of which are subclinical.

24   [46] P.R. Larsen, et al., "Thyroid Hypofunction Appearing as a
    Delayed Manifestation of Accidental Exposure to Radioactive Fall-
25 out in a Marshallese Population, Vol. 1 **Proceedings of the
    Symposium of the Late Biological Effects of Ionizing Radiation
26 held by the International Atomic Energy Agency** (1978).

27   [47] Assumedly meaning clinical hypothyroidism.

28 **ORDER RE SUMMARY JUDGMENT-   63**

1 Larsen found that 4 of the 43 Rongelapese had abnormally high

2 basal TSH and TRH (Thyrotropin Releasing Hormone)-induced TSH

3 release as opposed to only 2 of 214 controls. In three-quarters

4 of these subjects, the estimated thyroid exposure was less than

5 400 rads. Id.

6   Defendants contend Larsen's 1977 article contains "old" dose

7 estimates which are lower than more recent dose estimates devel-

8 oped using more sophisticated techniques. In Larsen, the esti-

9 mated mean dose for the exposed Rongelap population was 556 rads.

10 (Larsen at p. 104). However, a 1987 article by T. Hamilton

11 indicates the estimated mean dose for the Rongelapese was 2100

12 rads. (T. Hamilton, et al., "Thyroid Neoplasia in Marshall

13 Islanders Exposed to Nuclear Fallout," 258 **The Journal of the AMA**

14 629, 633).[48]

15   Defendants assert Mayer's reliance on Larsen is misplaced

16 for several additional reasons, including because Table IV of the

17 Larsen article indicates that 10 percent of the **unexposed control**

18 population had elevated TSH readings (versus 9 percent of the

19 **exposed Rongelapese**). (Larsen 1978 at p. 108). Defendants also

20 contend Mayer ignored data indicating that on Uritik island only

21 1 of 164 individuals (0.6 percent prevalence) developed

22 hypothyroidism, and the mean dose was 280 rads. (Maxon, et al.,

23 "Biologic Effects of Radioiodine on the Human Thyroid Gland,

24 **Werner and Ingbar's The Thyroid: A Fundamental and Clinical Text**

25 (Lewis E. Braverman and Robert D. Utiger, eds.) (1996) at pp.

26

---

27   [48] Defendants' Ex. 43.

28 **ORDER RE SUMMARY JUDGMENT-**   **64**

1 347-48).[49]

2 Finally, defendants say Mayer failed to take into account

3 that the Marshallese were exposed to a mixed fallout of different

4 kinds of radiation, including I-131. (Mayer Dep. at 246). They

5 cite deposition testimony from plaintiffs' expert Dr. Kelly

6 Clifton that "roughly two-thirds to 80 percent or 70 percent of

7 the dose may be from I-132, 33 and 35." (Clifton Dep. at p.

8 140). Clifton specifically rejected reliance on Marshall Island-

9 er data to support a dose-response calculation for I-131. In his

10 1995 report[50], Clifton stated the prophylactic thyroid hormone

11 treatment received by the exposed Rongelapese beginning in 1965

12 and by the Alingnae Marshallese in 1969 "invalidate[s] the use of

13 any data collected after its inception for quantitative estima-

14 tion of risk." (Clifton 1995 Report at p. 9).

15 In response to defendants' attack on Mayer's use of Larsen,

16 the best the plaintiffs can argue is that "even without the

17 Larsen data, [Mayer's] analysis remains essentially the same."

18 Mayer contends he could not ignore the Larsen results because he

19 could find no published errata or disclaimer for those results.

20 Subsequent to submission of his report, Mayer "refit" his model

21 without the Larsen data in order "[t]o be cautious and on the

22 firmest scientific grounds." According to Mayer his "results

23 were not greatly affected" because the doubling dose went to 60

24 _____

25 [49] Defendants' Ex. 78.

26 [50] "Carcinogenic Effects of Ionizing Radiation on the
   Thyroid Gland with Special Reference to Radioiodine and Thyroid
27 Cancer."

28 **ORDER RE SUMMARY JUDGMENT-   65**

rads, a value well within the uncertainty limits of the original

Weibull model." (Mayer Declaration at Paragraph 68).

Mayer explained why this was so:

> I picked a conservative error range for the
> doubling curve in recognition of the uncertainties
> and gaps in the individual studies. That is why
> adjusting the data to remove points, or move
> them around, as I have done in response to
> defendants' criticism, does not drive my
> doubling-dose estimate out of the error range.
> **As long as I retain one low dose study, e.g. Nagataki,
> my doubling-dose estimate is stable and robust
> within the uncertainty range stated.**

(Mayer Declaration at Paragraph 69) (Emphasis added).

Ultimately, all Mayer has to rely on is Nagataki. Mayer

asserts findings by Nagataki that low level radiation has a

statistically significant effect on the risk of developing

hypothyroidism is consistent with Larsen's findings for the

Marshallese "at radiation exposures of about 350 rads." However,

as noted above, there are serious shortcomings in Mayer's

interpretation of Nagataki. Furthermore, Mayer claims Nagataki

shows statistical significance at between 40 and 70 rads, figures

which are significantly lower than 350 rads.

(d) Maxon 1977[51]

In his report, Mayer cited Maxon 1977 for the proposition

that at an "approximate dose" of 100 rads the "approximate

prevalence" of hypothyroidism is 2.0 (2 percent). (Mayer Rpt. at

---

[51] Maxon, et al., "Ionizing Irradiation and the Induction
of Clinically Significant Disease in the Human Thyroid Gland," 63
The American Journal of Medicine 967 (Dec. 1977). (Defendants'
Ex. 77).

ORDER RE SUMMARY JUDGMENT-   66

p. 8).   Mayer acknowledged that Maxon makes no mention of such a

prevalence, but that he [Mayer] extrapolated the figure from

Maxon's data.   (Mayer Dep. at 202).   Mayer was unable to explain

during his deposition how he extrapolated to this prevalence

figure.   (Mayer Dep. at pp. 198-99; 203).

    According to defendants, Mayer most likely derived his

prevalence figure from Maxon's reference to some "preliminary

results" from a P. Hamilton[52]:

> Preliminary results of a follow-up survey of
> subjects regarded as having normal thyroids
> after diagnostic iodine-131 tests at ages
> of less than 16 years suggest that eight of
> 443 (1.8 percent) subsequently became hypo-
> thyroid . . . .

(Maxon 1977 at pp. 971-72).   Defendants contend Mayer's reliance

on these "preliminary results" is improper.   The results appar-

ently were never published nor is there any indication that

Hamilton produced any "final results."   Defendants argue that

Mayer cannot get any specific dose information from Maxon because

it is not provided by Maxon.   Defendants note Maxon is merely a

"review" piece, rather than an epidemiological study.   Therefore,

they argue that any information which does not originate with

Maxon, including dose, cannot be relied upon by Mayer because it

"is not available for critique and analysis."

    Defendants claim Mayer is selective in his use of Maxon and

ignores certain pertinent information contained therein, includ-

ing: 1) Hamilton's "preliminary data" which indicated no cases

---

[52]   Hamilton, P., et al., "Works in progress: diagnostic
radioiodine 131 in children, personal communication of prelimi-
nary results," (September 4, 1975).

ORDER RE SUMMARY JUDGMENT-   67

1   of hypothyroidism attributable to radiation in 146 patients

2   exposed to a mean dose of 18 rads; 2) Rallison's finding of only

3   two cases of **overt** hypothyroidism in 1,378 children exposed to I-

4   131 fallout, versus four cases in 3,801 non-irradiated control

5   subjects based on a mean dose exposure of 18 or 46 rads- a

6   difference which is not "statistically significant" (Maxon 1977

7   at p. 972); 3) Hempleman's finding that none of 105 patients who

8   had received a mean dose of 399 rads of external ionizing

9   radiation in early childhood was **clinically** hypothyroid; 4)

10 Refetoff's finding of no **clinical** hypothyroidism in 100 patients

11 exposed to incidental external irradiation of the thyroid,

12 seemingly less than 1,000 rads during childhood. (_Id_.at 969); 5)

13 Maxon's proposal of a linear model with a threshold "since a

14 large number of cells would probably have to be altered to result

15 in [hypothyroidism] due to the large functional reserve capacity

16 in the thyroid gland." (Maxon 1977 at p. 968). Defendants point

17 out Mayer's dose response curve assumes there is no threshold.

18 (Mayer Dep. at pp. 320-21).

19   Maxon stated that for the purposes of his study, the primary

20 criterion for determining thyroid hypofunction was **clinical**

21 **hypothyroidism** as diagnosed by reporting physicians. Maxon

22 indicated his report was concerned with "clinically evident

23 disease" and much of his data had been collected prior to the

24 availability of more sophisticated biochemical tests of thyroid

25 function. (Maxon 1977 at pp. 968-69). The Rallison, Hempleman

26 and Refetoff studies all refer to overt or clinical

27 hypothyroidism.

28 **ORDER RE SUMMARY JUDGMENT-   68**

In his declaration, Mayer says he "aggregat[ed] **clinical** and subclinical hypothyroidism data" to make his dose-response curve more accurate. He says that with the exception of Kaplan, all of the other data points involved either hypothyroidism, subclinical hypothyroidism, or some combination thereof and by using them all, he minimized the problem of different definitions between clinical and subclinical hypothyroidism. (Mayer Declaration at Paragraphs 61 and 62). Therefore, it is not clear why Mayer ignored the Rallison, Hempleman and Refetoff studies.

In his declaration, Mayer still does not say exactly how he derives from Maxon a doubling of risk for hypothyroidism at 100 rads. He says only that he "considered the full body of this scientist's work in coming up with a conservative risk of hypo-thyroidism at 100 rads." (Mayer Declaration at Paragraph 59). He asserts his use of Maxon benefits the defendants:

> I used the Maxon slope for hypothyroidism
> to try to make sure I included as much
> data as possible to help pin down the uncertainty
> in the region of greatest interest. This point
> actually lowers my curve and raises the doubling
> dose. Thus, inclusion of the Maxon data renders
> a more conservative result than that shown by
> Nagataki, et al. If defendants want me to remove
> if from my analysis, I will have to lower my
> doubling dose and narrow my uncertainty range.
> However, I believe the data point should be kept.

(Mayer Declaration at page 58).

Mayer once again retreats to sole reliance upon Nagataki.

(3) Biological Plausibility

Plaintiffs assert Dr. Mayer has a biological basis for his curve-fitting. However, at his deposition, Mayer was not at all

ORDER RE SUMMARY JUDGMENT-   **69**

1 certain about this. Mayer stated that at low doses, "it is [his]

2 **suspicion** that you get auto-immune disease leading at least to

3 subclinical hypothyroidism." He added that this was merely a

4 "hypothesis" or an assumption. (Mayer Dep. at pp. 175-

5 76)(Emphasis added).

6   When asked whether the Nagataki study showed an association

7 between hypothyroidism and radiation exposure of less than 1 gray

8 (100 rads), Mayer stated "[i]t lends evidence to an association,"

9 but [i]t will take many more studies." (Mayer Dep. at p. 275).

10 Said Mayer: "The sad part is, we haven't done enough studies to

11 have a definitive conclusion, but we certainly have evidence

12 leading strongly in that direction." (Id.) Elsewhere in his

13 deposition, Mayer testified the best he could do was a "rough

14 estimate" because of the "limited stage of knowledge and the

15 early stage of investigation . . . .," and because "[w]e know

16 very little about the relationship between radiation and

17 hypothyroid." (Mayer Dep. at p. 154).

18   Plaintiffs assert the data supporting the existence of, and

19 the biological basis for, low-dose delayed onset hypothyroidism

20 is established in the reports and depositions of their experts,

21 Drs. Peters, Clifton, Ruttenber and Radford.

22   Plaintiffs cite a passage from the report of Dr. Sara

23 Peters:

24   Malone and Cullen have described two mechanisms
            of radiation related hypothyroidism. Early
25   dysfunction, accounting for 5-40% of cases,
            appears to be dose-dependent and related to
26   thyroid follicular death [direct cell-killing].
            Late-onset hypothyroidism does not appear to be
27   dose-dependent and may be related to the presence

28 **ORDER RE SUMMARY JUDGMENT-**   **70**

of autoimmune factors.

(Peters Rpt. at p. 8).

Peters offers only speculation that late-onset hypothyroidism "may" be related to autoimmune factors. She does not represent that it is generally accepted that late-onset hypothyroidism "is" related to autoimmune factors or that radiation exposure, and particularly low dose exposure, is accountable for the autoimmune process.

Plaintiffs note that Dr. Clifton, in his report, cites a 1980 report titled "Irradiation and thyroid disease: Dosimetric, clinical and carcinogenic aspects," by Dumont, J.E., Malone, J.F., and Van Herle, A.J. A passage from the Dumont report states:

> Recent work demonstrates that the incidence of hypothyroidism **appears** to be a two-step process, one of which is dose related. The early incidence, two years after therapy . . . is a linear function of dose for both 131-I and 125-I. Therefore, the mechanism underlying it must be closely associated with the amount of radiation damage developed in the gland. The rate of incidence, that is the additional increment of hypothyroidism per year, from two years onwards, is relatively dose independent, indicating it is not strongly associated with primary radiation damage [direct cell-killing]. It is more likely to be determined by a fundamental biological process involving the gland itself and **may** be initiated by the radiation insult.

(Emphasis added).

This report is as equivocal as Dr. Peter's opinion. Furthermore, Dr. Clifton only cited the Dumont report. He did not quote the passage above and did not discuss in his report the biological basis for late-onset hypothyroidism.

Plaintiffs claim a 1996 report entitled "Health Consequences

ORDER RE SUMMARY JUDGMENT-   71

of the Chernobyl Accident: Results of the IPHECA Pilot Projects

and Related National Programmes" by Souchkevitch et al.[53], con-

firms that autoimmune responses are known to start at low exposu-

res, for example below 30 rads. Plaintiffs make this conclusory

statement without explaining the epidemiological analysis in the

report which allegedly supports it. They do not offer the

declaration of any expert attesting that the IPHECA report

reached such a conclusion.

The defendants set forth numerous and detailed reasons about

limitations in drawing conclusions from the IPHECA report. Dr.

Fred Mettler, Chairman of the Department of Radiology and Nuclear

Medicine at the University of New Mexico, was a member of the

committee that recommended the World Health Organization (WHO)

establish IPHECA (International Programme on the Health Effects

of the Chernobyl Accident). He cannot locate any finding in the

IPHECA report that low to moderate doses of radioiodine trigger

autoimmune thyroid conditions (including the release of thyroid

antibodies) or that Chernobyl emissions are responsible for

autoimmune thyroid disease in areas affected by Chernobyl emis-

sions. (Mettler Declaration at Paragraphs 36-38).[54] Indeed,

the IPHECA report indicates that while an increased level of

antibodies has been observed in exposed children versus the

control group, "it would be premature to interpret these

indicators as a manifestation of autoimmune thyroiditis."

_____

[53]   Defendants' Ex. 183.

[54]   Defendants' Ex. 196.

ORDER RE SUMMARY JUDGMENT-   72

(IPHECA 1996 Report at p. 267).

Plaintiffs say their expert Dr. Radford, at his deposition, articulated the biological logic for autoimmune reactions as the explanation for delayed onset hypothyroidism. (Radford Dep. at p. 357). However, the best Radford could offer was a hypothesis as to how radiation at low doses "may" influence hypothyroidism. According to him, "a body of scientific information is developing which does **suggest** a relationship between autoimmune thyroid disease and radiation exposure." The relationship is not "established," however, and "warrants further study." (Radford Dep. at p. 432) (Emphasis added).

Dr. Ruttenber offers no better:

> I think . . . there is evidence that suggests now that autoimmune thyroid disease . . . **could** be caused by radiation, and there is evidence that the doses which cause autoimmune disease are . . . lower than the doses that can cause what might be called clinical hypothyroidism . . . and then clearly lower than the clinical hypothyroidism that's caused by tissue damage to the thyroid.

(Ruttenber Dep. at pp. 74-75) (Emphasis added).

Plaintiffs have submitted a declaration from Eric Gershwin, M.D., an immunologist at the University School of Medicine at Davis. (Ex. 1 to Plaintiffs' Appendix 1 re Iodine Claims).[55] **According to plaintiffs**, Dr. Gershwin concludes the dose-response relationship between low-level radiation exposure and hypothyroidism described in Mayer's analysis "'is not only consistent, but, more importantly, is what one would predict,'

---

[55] Dr. Gershwin did not prepare an expert report on behalf of the plaintiffs.

ORDER RE SUMMARY JUDGMENT-   73

1 given the **state of knowledge** concerning autoimmune mechanisms."

2 (Emphasis added).

3   Gershwin states:

    Autoimmune thyroiditis is well known in both
    humans and animals. In the case of thyroiditis
    at Hanford, the radiation would release antigen
    from the thyroid as a result of the radiation
    injury. There have already been large numbers
    of studies which have indicated that susceptibility
    to autoimmune thyroiditis are linked to MHC Class
    II genes. In addition, autoimmune thyroiditis can
    be induced by combination of methodologies including
    immune suppression and irradiation of animals.
    There are also influences to other genes including
    MHC Class I and/or non-MHC genes which influence
    the severity and incidence of thyroiditis. Thus,
    the release of antigen following radiation exposure
    at Hanford would lead to an immune response that would
    vary greatly and therefore, reflect different
    susceptibilities depending on the genetics (DNA) of the
    exposed person.

    Hence, the experience with the Atomic bomb, as
    reflected in the study by Nagataki, et al., and
    the Chernobyl accident, as reflected in the WHO
    materials on microsomal autoantibodies, is not
    only consistent, but, more importantly, is what
    one would predict.

17 (Gershwin Declaration at Paragraphs 8 and 9).

18   Dr. Gershwin says nothing about **Mayer's methodology** and

19 probably could not do so since Gershwin is not a biostatistician.

20 Furthermore, Gershwin does not say **Mayer's work** is "consistent"

21 with "the state of knowledge concerning autoimmune mechanisms."

22 There is no endorsement of Mayer's work. Gershwin opines that

23 the Nagataki and Chernobyl data are "consistent" with knowledge

24 concerning autoimmune mechanisms. However, Gershwin does not

25 explicitly opine that **low to moderate** radiation exposure can lead

26 to an autoimmune response, or specifically that it can lead to

27 "autoimmune hypothyroidism." Gershwin limits his comments to

28 **ORDER RE SUMMARY JUDGMENT-**   **74**

"autoimmune thyroiditis." His use of the phrase, "state of the knowledge" implies there are limitations in the knowledge of the relationship between low-dose radiation and hypothyroidism. This is borne out by the Nagataki and Chernobyl data.

According to the declarations of Dr. Mettler and Dr. Robert Anderson, a radiation pathologist and Professor of Pathology at the University of Minnesota Medical School[56], the scientific community has only hypothesized about possible associations between autoimmune thyroid conditions and low to moderate dose radiation; and has only theorized about the underlying biological mechanism involved. (Anderson Declaration at Paragraph 10; Mettler Declaration at Paragraph 12). Anderson says he is not aware of any study in the peer reviewed scientific literature demonstrating that low to moderate dose radiation to the thyroid triggers an autoimmune response. (Anderson Declaration at Paragraph 9). The 1993 Hanford Thyroid Disease Study (Study Protocol) states that "[w]hile partial or complete hypothyroidism may occur at lower doses, it is generally accepted that 1500-2000 rad from external gamma radiation is a threshold above which all persons become hypothyroid." (Study Protocol at p. 6).[57]

(4) "Biostatistical" Methods

The plaintiffs contend Dr. Mayer applied standard biostatis-tical methods to his analysis of the dose-response relationship

_____

[56] Anderson Declaration, Defendants' Ex. 199.

[57] Defendants' Ex. 51.

ORDER RE SUMMARY JUDGMENT-   75

1 between radiation and hypothyroidism. Mayer says his purpose in

2 this case was to "develop, test and apply methods and models for

3 the analysis of observational data." Mayer emphasizes he has no

4 control over the observational data. (Mayer Declaration at

5 Paragraph 11).

6   The plaintiffs apparently recognized the limitations of the

7 epidemiological data relating to low dose radiation and

8 hypothyroidism and hence, that is why Mayer was hired. According

9 to plaintiffs, "when published findings are limited, it is

10 standard practice in biostatistics and epidemiology, as in all

11 scientific disciplines, **to make assumptions and extrapolations**

12 **based on the published literature extant**." The plaintiffs claim

13 this is "well-established exercise of scientific expert

14 judgment."

15   Plaintiffs say Mayer's approach is consistent with the

16 Hanford Environmental Dose Reconstruction (HEDR) approach "in

17 establishing parameters and setting parameter values for its dose

18 reconstruction models." Plaintiffs point out several examples of

19 HEDR assuming parameter values because of the lack of underlying

20 data, or because of suspicions about the reliability of

21 underlying data.

22   Plaintiffs claim Mayer did the same thing, one example of

23 which is his derivation of "standardized prevalence" rates.

24 Mayer says he derived prevalence rates by standardizing crudely

25 to the population and asserts this is a recognized biostatistical

26

27

28 **ORDER RE SUMMARY JUDGMENT-   76**

1 method for reducing distortion in comparing populations.[58]

2 Another example, say plaintiffs, is Mayer's fitting a curve to

3 data points and his uncertainty analysis of the doubling dose

4 estimate. Plaintiffs claim this is similar to curve-fitting

5 techniques and uncertainty analysis employed by HEDR.

6   Plaintiffs assert Mayer's "grouping" of data is part of

7 standard biostatistical methodology (i.e. grouping of data

8 pertaining to different diseases, etc.). As support, plaintiffs

9 cite testimony from Dr. Ruttenber. However, all Ruttenber stated

10 was that he was not "interested" in or "asked" to do the

11 "comparative scaling" done by Mayer. Ruttenber said he was not

12 aware of a biostatistician doing what Mayer did, although he was

13 aware of one study which combined malignant and benign neoplasms

14 in assessing dose-response. (Ruttenber Dep. at p. 203).

15 Ruttenber testified that he did not feel "that grouping thyroid

16 diseases [was] a useful thing in determining exposure and disease

17 relations." (Ruttenber Dep. at p. 172).

18   As further support for Mayer's grouping of data "focusing on

19 the broader class of thyroid disease," plaintiffs cite some

20 caselaw and a brief passage from a general reference text on

21 epidemiology (Lilienfeld & Lilienfeld, <u>Foundations of</u>

22 <u>Epidemiology</u>). However, those sources do not focus on the

23 _____

24   [58] Mayer selected a 1 percent background prevalence rate.
He claims this is a sound selection for the general population

25 and "is widely cited in the articles and texts on thyroid diseas-
es written for endocrinologists." Mayer acknowledges there are

26 differences in prevalence due to age and gender, but asserts it
is inconsequential for his relative risk calculation. (Mayer

27 Declaration at Paragraphs 23 and 24).

28 **ORDER RE SUMMARY JUDGMENT-**   **77**

1 specific issue here: whether Mayer's grouping of thyroid disease

2 data in this case is scientifically supportable.

3   The defendants claim Mayer did not use standard

4 biostatistical methods. Indeed, there is a portion of Mayer's

5 deposition testimony which confirms as much. It also explains

6 why plaintiffs argue Mayer's method is similar to HEDR's method

7 and therefore should be considered scientifically valid:

8   **A: The conversions of rates and things like
       that, these are not what I standardly do in**

9   **statistics. One of the places I got this idea
       was from reading the HEDR model and the methodology**

10   **being used here that this area seemed to be an
       area where people convert things by units, by**

11   **equivalency factors and all that. So I didn't
       really think anything of it.**

12
       Q: Can you tell me what it is in the HEDR

13   model or HEDR methodology that is comparable
       to taking data concerning a threshold dose and

14   converting it as you did?

15   A: My humble opinion of HEDR, the pieces I have read,
       it has nothing to do with dose, but they make up

16   numbers all the time. They even admit to making
       up numbers, taking medians or transferring numbers,

17   because we don't have enough data to do formal
       statistical analysis, and this is standard modeling

18   methodology.

19   Every piece of HEDR and review of HEDR I have
       read and a seminar that I attended by members of the

20   HEDR committee discussed this modeling methodology.
       You try to get comparable numbers, and if you can't,

21   you use the best numbers you have. I am a
       statistician, and the amount of missing data is

22   astronomical, and they do the best, they approximate
       it, they put in zeroes or they take averages, they

23   extrapolate or interpolate. And they talk about it
       quite openly.

24
       Now they do provide appendices, usually, but they are

25   [a] little bigger operation than I am. I am a one-man
       band, one-person band. **But I did try to conform to**

26   **what I saw as methodology within what I would call the
       radiation modeling literature. . . .**

27

28 **ORDER RE SUMMARY JUDGMENT**-   78

> **Really good data is not available. You work with**
> **what you have, and you don't mind transforming it,**
> **if the dose information is inaccurate, the name, the**
> **categorization of the diseases is highly inaccurate.**
> **One article talks about subclinical in one definition;**
> **one talks about subclinical with another.**
>
> So given all those errors, you do the best you can.
> That's why you put error bounds on it and try to
> have an upper and lower bound.

(Mayer Dep. at pp. 211-213) (Emphasis added).[59]

Plaintiffs do not attempt to defend Mayer's methodology by citing to "to some objective source [in biostatistics]- a learned treatise, the policy statement of a professional association, a published article in a reputable scientific journal or the like- to show [he has] followed the scientific method." Daubert II, 43 F.3d at 1319. Compounding the problem is that Mayer admits he is on new turf with regard to radiation modeling, an area with which he has no prior experience. Therefore, he essentially tries to emulate or guess HEDR's approach. This is where Mayer's qualifications become critical, as is discussed infra.

With regard to the "grouping of data," defendants argue Mayer did this simply as a matter of expediency, not because it is standard practice in biostatistics. At his deposition, Mayer was asked if he was aware of Dr. Ruttenber's statement that hypothyroidism caused by autoimmune thyroiditis should be treated separately from non-autoimmune hypothyroidism. Mayer's response was:

---

[59]   Elsewhere in his deposition, Mayer stated his curve-fitting represents **"non-statistical** statements of uncertainty." (Mayer Dep. at 315). And of course, in his report, he conceded there is not enough data to fit a formal statistical analysis. (Mayer Rpt. at p. 5).

ORDER RE SUMMARY JUDGMENT-   79

1
2
3
4
5

> This goes back to the problem of lumping
> and splitting. In this area, I tend to
> be more of a lumper, and he tends to be
> more of a splitter. And if I had enough
> data, and adequate data, I would split the
> data down by a lot of categories, a lot more
> than just those categories. The fact of the
> matter is, I could not split the data and
> have enough data- I had no way to do that.

6   (Mayer Dep. at p. 172).

7   Mayer said he agreed with Ruttenber "that there are two

8   different processes going on" and that splitting by etiology

9   should be done. (Mayer Dep. at pp. 173-74).[60] However, he

10 excused his failure to split by etiology on the basis that there

11 was not enough data, and because "it is **probably** not true that at

12 some dosage, you quit getting one, and you start getting

13 another." According to Mayer, [t]here is **probably** a slow

14 transition from one into the other, meaning the low dose, [and]

15 it is my **suspicion** that you get auto-immune disease leading at

16 least to subclinical hypothyroidism." Mayer referred to this as

17 a "first order of approximation," or "rough first order analysis"

18 of the data, "treat[ing] them [autoimmune hypothyroidism and non-

19 autoimmune hypothyroidism] as one." (Mayer Dep. at pp. 174 and

20 178)(Emphasis added). The fundamental problem, however, is that

21 a mere "suspicion" is not enough to justify Mayer's grouping of

22 ────────────────

23 [60] Mayer stated it was important for him to try to find out
what definitions of hypothyroidism were being used in the
epidemiological studies examined by him. He stated it was

24 important for him to know the categorization "because two people
can use the category 'subclinical' and mean quite different

25 things." This is so, according to Mayer, because it can impact
the analysis of prevalence rates. (Mayer Dep. at pp. 92-93). He

26 went on to say that "the type of pooling that people do in this
kind of data is very dangerous when you have different

27 definitions, different statistics." (Mayer Dep. at p. 316).

28 **ORDER RE SUMMARY JUDGMENT-   80**

1   data.

2   Defendants contend that after his deposition, but before he

3   produced his computer files, Mayer secretly changed the formula

4   he used to generate the dose-response curve in his report.

5   Defendants say the purpose of this was so he could correct

6   fundamental flaws in his methodology. Defendants note that

7   Mayer's report does not explain how he obtained his

8   hypothyroidism prevalence ratios. Following his deposition,

9   Mayer prepared a document which plaintiffs' counsel sent to

10   defense counsel. This document, "Response to Deposition Inquiry

11   Concerning Conversion and Standardization of Epidemiological

12   Measures of Effect,"[61] purports to explain how Mayer obtained

13   his prevalence values.

14   Defendants argue the document represents an attempt by Mayer

15   to shore up his analysis and cure a less than stellar deposition

16   performance. Defendants say it is "new work product to explain

17   undocumented adjustments that he allegedly made one year earlier

18   [and] contemporaneously with the preparation of his report." In

19   other words, defendants contend Mayer, at the time of his

20   deposition, had no work product which had been prepared

21   contemporaneously with his report.

22   Plaintiffs say Mayer's "Response to Deposition Inquiry

23   Concerning Conversion and Standardization of Epidemiological

24   Measures of Effect," was merely an attempt to assist defendants

25   in understanding how he obtained the prevalence values.

26   _____

27   [61]   Defendants' Ex. 197.

28   **ORDER RE SUMMARY JUDGMENT-   81**

1   According to plaintiffs, the computer spreadsheet they provided

2   to defendants (at the same time they provided the aforementioned

3   document) was prepared prior to the submission of Mayer's report.

4   Defendants contend the computer spreadsheets supplied to

5   them contain a dose-response model different "in several material

6   respects" from the model and curve-fitting formula contained in

7   Mayer's November 1995 report. Defendants go into great detail

8   explaining these differences (i.e. spreadsheet indicates a 3-

9   parameter Weibull curve was used, whereas report says a 2-

10 parameter Weibull curve was used) and offer a declaration from

11 Dr. M. Laurentius Marais[62], to verify the differences.[63]

12   Dr. Marais says the change in formulas corrects a

13 fundamental flaw in the original formula contained in Mayer's

14 report. Marais asserts the original formula predicts a zero

15 percent prevalence of hypothyroidism at a dose of 1 rad.

16 According to Marais, if the model predicts a prevalence of zero

17 at one rad and below, such that even the general population does

18 not have hypothyroidism, then by definition, the risk can never

19 be doubled at any dose. Defendants therefore claim Mayer changed

20 his model "to fix a basic flaw that reflected poorly on his

21 methodology."

22   Certainly, **if Mayer altered his model in material respects**

23 _____

24   [62] Marais is a vice-president and senior consultant in a
    consulting firm specializing in applied mathematics and
25 statistics, including the statistical analysis of epidemiological
    data. He has a Ph.D. from Stanford in mathematics and
26 statistics.

27   [63] Defendants' Ex. 206.

28 **ORDER RE SUMMARY JUDGMENT-   82**

1 **without informing the defendants,** that reflects poorly on his

2 credibility and the reliability of his analysis. However, the

3 court deems it unnecessary to determine whether or not these

4 alleged statistical machinations occurred. The critical issue is

5 if the epidemiological data supports Mayer's analysis, whether

6 that is the analysis set forth in his original report, or the

7 alleged revised analysis set forth in the computer spreadsheet,

8 together which conclude the risk of hypothyroidism doubles at a

9 low dose range, whether that is 23-87 rads, 25-89 rads, or 30-80

10 rads.

11   The court believes Mayer has drawn inferences from the

12 epidemiological data which are scientifically unreliable and

13 methodologically unsound.

14

15   **c. Qualifications**

16   Statistics is the science and art of gaining information

17 from data. For statistical purposes, data means observations and

18 measurements expressed as numbers. Federal Judicial Center

19 <u>Reference Manual on Scientific Evidence</u>, "Reference Guide on

20 Statistics" (1994) at p. 335. Specializations such as

21 biostatistics are primarily statistical, with an emphasis on

22 methods and problems most important to the related substantive

23 discipline. <u>Id</u>. at 336. According to the "Reference Guide on

24 Statistics:"

25   Experience with applied statistics is
            the best indication of the type of statistical
26   experience needed in court. By and large,
            individuals who think of themselves as
27   specialists in using statistical methods-

28 **ORDER RE SUMMARY JUDGMENT-   83**

1   and whose professional careers demonstrate
           this orientation- are most likely to apply
2   appropriate procedures and correctly interpret
           the results. **At the same time, the choice of**
3   **which data to examine or how to best model a**
           particular process may require subject matter
4   **expertise that a statistician may lack.**
           Statisticians typically advise experts in
5   **substantive fields on the procedures for**
           collecting data and usually analyze data collected
6   **by others. As a result, cases involving statistical**
           evidence often are (or should be) 'two expert'
7   **cases of interlocking testimony. . . . [T]he**
           value of the statistical analysis depends on
8   **the substantive . . . knowledge that informs it.**

9   Id. at 336-37 (Emphasis added).

10   Statistical analysis is only as good as the data upon which

11   it is based. Consequently, it is important to verify the quality

12   of the data and identify its limitations. Id. at 341. Mayer's

13   "statistical" analysis, (if that is indeed what it is in light of

14   his statements that he could not conduct a "formal" statistical

15   analysis), is based upon data consisting of observational studies

16   of causation (epidemiological studies). Experts may disagree

17   about the value of certain observational studies, but "[i]n the

18   end, **deciding whether associations are causal is not a matter of**

19   **statistics, but a matter of good scientific judgment.**" Id. at

20   352 (Emphasis added).

21   Where a single witness presents both the substantive

22   underpinnings and the statistical analysis, ideally he should

23   have extensive experience in both fields. Less may suffice to

24   qualify the witness under FRE 702. Qualifications in one field

25   do not necessarily imply qualifications in another. Id. at 337,

26   n. 4.

27   The plaintiffs assert Mayer is qualified to render the

28   **ORDER RE SUMMARY JUDGMENT-   84**

1 opinions expressed by him because he is "an extremely well-

2 credentialed biostatistician with clinical training." Plaintiffs

3 concede Mayer makes no claim to be an expert in the field of

4 endocrinology or radiation health effects, but that he is an

5 expert in biostatistical analysis of health-related data, the

6 fitting of biostatistical models, and the making of statistical

7 inferences. Plaintiffs acknowledge it is "important" that as a

8 biostatistician, Mayer be "unusually well-trained and

9 knowledgeable in clinical medicine and medical research,

10 including such areas as thyroid disease and radiation health

11 effects."

12   Defendants do not quibble with Mayer's statistical

13 expertise. They say Mayer arguably could have provided

14 statistical support to credentialed experts in radiation health

15 effects and thyroid disease. What they say is problematic is

16 that Mayer has no education, training or experience in radiation,

17 thyroid disease, or any related field and that this is material

18 because Mayer's opinion focuses solely on the relationship

19 between radiation and thyroid disease, and "relies on careful

20 reading of the medical and epidemiological literature on the

21 effects of radiation on the thyroid." (Mayer Rpt. at p. 5).

22   Mayer has never had a clinical practice. (Mayer Dep. at pp.

23 33 and 378).[64] In support of their claim that Mayer is

24 "unusually well-trained and knowledgeable in clinical medicine

25 _____

26   [64] Mayer testified he knew he was going to be a researcher
    and not a clinician. (Mayer Dep. at p. 376).

27

28 **ORDER RE SUMMARY JUDGMENT-   85**

1   and medical disease, including such areas as thyroid disease and

2   radiation health effects," the plaintiffs offer passages from

3   Mayer's deposition testimony, including a vague assertion that he

4   has been "involved in the design or comment of studies involving

5   radiation in a clinical setting, but only as a methodologist or

6   biostatistician." According to Mayer, this activity entails

7   being "asked questions from time to time on the design or

8   carrying" out of studies of cancer in adults. (Mayer Dep. at p.

9   349). This is not evidence of substantive involvement by Mayer

10 in any area relating to radiation or radiation health effects,

11 and particularly thyroid disease.

12   The work Mayer was asked to perform here should not and

13 could not have been done without experience in the fields which

14 are the subject of his biostatistical analysis. Mayer did not

15 simply extract data and crunch numbers for the use of persons

16 with expertise in thyroid disease or health effects, but

17 purported to interpret studies that require substantive knowledge

18 about radiation biology and thyroid disease etiology.

19   It is clear that Mayer does not possess the level of

20 substantive knowledge in these areas sufficient to qualify him as

21 an expert capable of drawing scientifically reliable conclusions

22 about the causal association between radioiodine exposure and

23 autoimmune hypothyroidism. His lack of qualification is

24 manifested in the unsoundness of his methodology, particularly by

25 the scientifically unreliable inferences he draws from underlying

26 epidemiological data. He is not qualified to interpret or apply

27 the studies that form the basis of his dose-response curve.

28 **ORDER RE SUMMARY JUDGMENT-   86**

d.   Summary and Conclusion

Dr. Mayer's statistical analysis cannot be divorced from the epidemiological studies on which it is based.[65]   Based on the discussion set forth above, the court concludes: 1) those studies do not support Dr. Mayer's "statistical analysis"[66]; and 2) Dr. Mayer is not qualified to render an opinion about the relationship between radiation and autoimmune hypothyroidism. Consequently, Dr. Mayer's statistical analysis is not "reliable" under the first prong of Daubert and it cannot assist the trier of fact in determining any fact in issue.

The shortcomings of the four low dose studies (Kaplan, Nagataki, Larsen and Maxon) in establishing an association between **low dose** radiation and autoimmune hypothyroidism are adequately detailed above.   Especially persuasive is the

---

[65] Dr. Anderson puts it this way: "Mathematical assessment of inappropriately utilized data does not render the data any less appropriate to the issue at hand."   (Anderson Declaration at Paragraph 24).   He adds that "[i]t is not possible to select an appropriate mathematical model for analyzing a dose-response relationship without having an understanding of the radiobiology at issue."   (Anderson Declaration at Paragraph 12).

[66] O'Connor v. Commonwealth Edison Co., 13 F.3d 1090, 1106 (7th Cir. 1994) (expert's method of diagnosis and his conclusion regarding causation were not supported by the authors on which he claimed to rely); Hall v. Baxter Healthcare Corp., 947 F.Supp. 1387, 1411 (D. Or. 1996) (expert made too great a leap from the underlying data to his conclusions and therefore, conclusions were not the result of the faithful application of valid scientific methodology); Muzzey v. Kerr-McGee Chemical Corp., 921 F.Supp. 511 (N.D. Ill. 1996) (proposition that radiation could cause polycythemia vera was not empirically proven and although epidemiological studies were not necessary to prove causation, the lack of any conclusive studies on the subject weighed against admissibility of that proposition).

ORDER RE SUMMARY JUDGMENT-   87

acknowledgement of the Kaplan study authors that their own study

does not establish a causal link between radiation and autoimmune

hypothyroidism.   See <u>Muzzey v. Kerr-McGee Chemical Corp.</u>, 921

F.Supp. 511, 519 (N.D. Ill. 1996).   Mayer cannot make up for this

lack of scientific support because as a threshold matter, he does

not have the necessary expertise in radiation health effects and

thyroid disease processes which would qualify him to interpret

the data (the epidemiological studies) in the manner which he

seeks.[67]

A number of other factors reinforce the conclusion that

Mayer's statistical analysis is unreliable.   There is no question

that he formed the opinions in his report solely for the purpose

of litigation and secondly, he never submitted his analysis for

publication and it has not been subjected to scientific peer

review.   Although those factors by themselves do not per se

warrant exclusion of Mayer's report, Mayer fails to adequately

---

[67]   An interesting comparison is <u>McCullock v. H.B. Fuller
Co.</u>, 61 F.3d 1038, 1043-44 (2nd Cir. 1995).   In that case, it was
argued that a medical doctor could not opine that glue fumes
caused throat polyps where the doctor was unable to point to a
single piece of medical literature standing for that proposition.
The court was unpersuaded because the doctor based his opinion on
a range of other factors including his care and treatment of the
plaintiff; the medical history of the plaintiff; pathological
studies; use of scientific analysis known as differential
etiology; and reference to various scientific and medical
treatises.   In other words, the doctor had other "substantive"
bases for his opinion.
Dr. Mayer has nothing to fall back on but numbers.   These
numbers are irrelevant without the necessary "substantive
underpinnings."   Lest there be any doubt that Mayer interpreted
the data, he testified in his deposition that "I tried to
standardize these studies as if they were one similar study,
**using my understanding of the biological mechanisms in
reinterpreting the data."**   (Mayer Dep. at p. 188) (Emphasis
added).

ORDER RE SUMMARY JUDGMENT-   88

1 explain how he reached his conclusions, or to point to an

2 objective source which supports the specific methodology employed

3 by him in this case.

4   Furthermore, it is not a "generally accepted" theory in the

5 scientific community that low to moderate dose radiation causes

6 autoimmune hypothyroidism. The plaintiffs cite a portion of Dr.

7 Ruttenber's testimony which they suggest is supportive of Mayer's

8 dose-response curve:

> Let me say one thing about Mayer's report
> at least, you know, looking at his dose-response
> analysis, it seems like that-- in terms of the
> spectrum of doses and biological effect that
> . . . at least from my quick reading of it, that
> the studies show slight effect with antibody
> positivity and thyroid disease, and then the
> ones that show higher-dose effects in terms of
> tissue damage-- I mean kind of line up on that
> curve. So it kind of summarizes that spectrum
> . . . and its consistent with the biologic ideas
> about causing hypothyroidism.

(Ruttenber Dep. at 205).

This is not a whole-hearted endorsement of Mayer's analysis

and even less so based on other comments made by Ruttenber:

> From what I understand of what he [Mayer] did, he
> is . . . attempting . . . to look at dose
> response for, essentially, **a lot of different
> types of hypothyroidism**, and he's attempting
> to look at dose response in . . . lower
> dose range than . . . I've looked at in the
> literature. [S]ome of the modeling that he
> did I don't understand completely, and I . . .
> haven't really formed an opinion on . . . how
> good it is or how bad it is. You know, I haven't
> looked at it in that much detail.

(Ruttenber Dep. at p. 202) (Emphasis added). Significantly, it

does not appear Ruttenber understood Mayer to be limiting himself

to autoimmune hypothyroidism.

ORDER RE SUMMARY JUDGMENT-   89

Plaintiffs cite to a portion of Dr. Radford's deposition as evidence that he endorsed Mayer's dose response curve:

> Well, I am just commenting on the extent to which you can draw a conclusion about the prevalence of hypothyroidism as a function of radiation dose. And the answer is, I think you can, and I think Doctor Mayer has done a reasonable job. There is scatter in the data, especially at high doses, but the data at the low doses seems to fit reasonably well, and therefore, I think it is a reasonable indication of a dose response curve for hypothyroidism.

(Radford Dep. at p. 467). However, Radford clearly recognized the limitations of the underlying epidemiological studies upon which Mayer relied. Radford acknowledged he had not personally reviewed those studies to determine whether the lines on Mayer's graph were properly plotted and although he had reviewed some of the **high dose** studies, he was "not so familiar" with the **low dose** studies. Radford stated Mayer's graph was "not a terribly good fit, but it is the best fit you can do with the range of data that exist." (Id. at p. 536). Radford indicated he had not done any type of analysis on Mayer's graph to see if it could withstand peer review. (Id. at p. 537).

Finally, Mayer's numerous methodological fluctuations indicate he is more concerned with the result than the science. In re TMI Litigation II, 922 F.Supp. 997, 1015 (M.D. Pa. 1996) (axiomatic that methodological fluctuations are not scientific). Under criticism from the defendants for his report's use of the Kaplan study, Mayer concludes he does not need it. Mayer also offers little defense for his report's use of Larsen and Maxon and ultimately is willing to forsake reliance on those studies.

ORDER RE SUMMARY JUDGMENT-   90

1 Now, following completion of his report and his deposition, he

2 asserts that all his model requires is the Nagataki study.

3 (Mayer Declaration at Paragraph 69). However, even that report

4 has some serious shortcomings and the defendants make a very

5 valid point: if using Nagataki gets Mayer a doubling dose that

6 is close to the dose at which Nagataki purportedly reports a

7 doubling of the risk, what exactly has Mayer added to the study?

8 Mayer essentially attempts to make something out of nothing.

9   Mayer's declaration is inconsistent in many respects with

10 his report and his deposition testimony.[68] For example, in his

11 report, Mayer said there was not enough data to fit a formal

12 statistical analysis complete with prediction intervals,

13 confidence intervals and hypothesis tests (Mayer Report at p. 5),

14 but in his declaration, shortcomings in the data are not a

15 problem because of the use of error ranges, confidence intervals

16 and point estimates. (Mayer Declaration at Paragraph 69). At

17 his deposition, he conceded he was not an expert in autoimmune

18 thyroid disease and that it was merely his "suspicion" that low

19 dose radiation exposure leads to autoimmune disease which, in

20 turn, leads to subclinical hypothyroidism. (Mayer Dep. at pp.

21 164-65; 174). However, in his declaration, he confidently

22 asserts the medical literature demonstrates there is an

23 association between radioiodine and hypothyroidism at doses as

24 low as 30 rads and that the biological basis for the association

25

26   [68] Fed. R. Civ. P. 26(a)(2)(B) requires the expert's report
   contain a **complete** statement of **all** opinions to be expressed and
   the basis and reasons therefor and the data or other information
27 considered by the witness in forming the opinions.

28 **ORDER RE SUMMARY JUDGMENT-   91**

is clear.   (Mayer Declaration at Paragraphs 34 and 47).

In the **four pages** of Mayer's report dedicated to his "Best estimate of the dose response relationship for radiation and hypothyroidism," there is no reference to autoimmune hypothyroidism.   There is no distinction between autoimmune and non-autoimmune hypothyroidism.   Plaintiffs have not offered a satisfactory explanation for this.   It was not until his subsequent deposition that Mayer made the distinction for the first time.   A significant portion of his **thirty page declaration**, submitted in response to the motion in limine, is dedicated to explaining the distinction.   In the process, Mayer cites numerous references which are not found in his report.

Whereas in his deposition Mayer only suspects that low dose radiation exposure causes autoimmune hypothyroidism, his declaration states without hesitation that it is so caused. Whereas in his deposition, Mayer concedes he is not an expert on the autoimmune process, his declaration attempts to give the impression that he is.   Mayer is a "moving target."

For all of the foregoing reasons, the court will grant defendants' motion in limine and exclude Dr. Mayer's report. Without Mayer, plaintiffs have no evidence on the pertinent summary judgment inquiry which is "at what radioiodine dose does the risk of autoimmune hypothyroidism double?"   Indeed, Mayer's analysis is not even sufficient to raise a genuine issue of material fact that radioiodine exposure **at low to moderate doses** is "capable of causing" autoimmune hypothyroidism (either starting the autoimmune process or pushing someone with an

ORDER RE SUMMARY JUDGMENT-   92

existing autoimmune disorder into clinical hypothyroidism).[69]

2.   A. James Ruttenber

a.   Introduction

Dr. Ruttenber prepared a report in 1995 entitled "Report of A. James Ruttenber, Ph.D., M.D., Regarding Causal Association Between Exposure to Iodine-131 from Hanford and Thyroid Disease." He addresses a number of non-neoplastic thyroid conditions, including chronic thyroiditis, also known as "autoimmune thyroiditis" and "Hashimoto's thyroiditis." It is his opinion about chronic thyroiditis which defendants challenge on <u>Daubert</u> grounds.

The defendants acknowledge Dr. Ruttenber is a "credentialed epidemiologist." They do not dispute his qualification to render

---

[69] Plaintiffs previously filed a motion for leave to file surreply or in the alternative to strike portions of defendants' reply regarding Dr. Mayer, in particular certain affidavits and declarations.
As is evident, the court finds the affidavit of Dr. Boice and the declarations of Dr. Kaplan and Dr. Howe properly address various assertions found in Mayer's lengthy declaration (i.e. progression theory) submitted as part of plaintiffs' response. The declaration of Dr. Mettler properly addresses arguments made by plaintiffs in their response concerning the IPHECA report. Dr. Mettler's declaration, as well as the declaration of Dr. Anderson, properly address assertions found in Mayer's declaration regarding the purported biological connection between low to moderate radiation dose exposure and autoimmune hypothyroidism.
All of these affidavits and declarations constitute legitimate responses to issue raised by plaintiffs in their response, a response which includes not only Mayer's declaration, but the declaration of Dr. Gershwin who had not previously submitted an expert report in this case. The court finds no basis for striking the affidavits and declarations submitted by defendants. The affidavit from Dr. Marais is not material to the court's decision and hence, the court makes no determination as to its propriety.

ORDER RE SUMMARY JUDGMENT-   93

an opinion about the causal association between I-131 exposure

and chronic thyroiditis. They do, however, dispute whether his

opinion is relevant to or "fits" the plaintiffs' generic

causation burden and secondly, whether his opinion is derived by

sound scientific methodology.

Ruttenber describes chronic thyroiditis as a general term

for chronic inflammation of the thyroid. It may result in

clinical or biochemical hypothyroidism and is most commonly

caused by autoimmune processes. He refers to four studies-

Spitalnik, Kaplan, Nagataki, and Kotani- which he says have

"linked" chronic thyroiditis to ionizing radiation exposure.

According to Ruttenber:

> . . . there is epidemiologic evidence
> that ionizing radiation produced a
> doubling of disease rates for autoimmune
> thyroiditis in exposed human populations.
> These studies satisfy the epidemiologic
> criteria for a **causal relation** between
> ionizing radiation and autoimmune thyroiditis.
> That is, there is consistency between results
> from studies of different populations, there
> is biological plausibility for the agent
> causing such damage to the thyroid, and the
> timing of the onset of disease in relation
> to the exposure is consistent with proposed
> biologic mechanisms.

(1995 Ruttenber Iodine Rpt. at pp. 16-17)(Emphasis added).

b.   **Fit/Relevancy**

Dr. Ruttenber does not offer an opinion about the dose of I-

131 (radioiodine) necessary to double the risk of contracting

chronic thyroiditis. Ruttenber and the plaintiffs acknowledge as

much. However, plaintiffs contend their burden is only to offer

ORDER RE SUMMARY JUDGMENT-   94

1   evidence that radioiodine exposure is "capable of causing"

2   thyroiditis at a certain dose range and that they were exposed to

3   that range of doses.   Plaintiffs say Ruttenber's report satisfies

4   this burden.

5   In his post-report and post-deposition declaration (Ex. 8 to

6   Plaintiffs' Appendix 1 re Iodine Claims), Dr. Ruttenber asserts

7   that "doubling doses are not necessary for causation."   He says:

8   In any case, I cite studies with doses in
             the range reported for Hanford exposure at
9   which disease rates are doubled.   I do not
             believe it is my responsibility to decide
10   exactly which of these studies is best or to
             average them or to try and fit them to a curve.
11   I view the Nagataki and Kaplan studies as
             providing **part** of the evidence that autoimmune
12   thyroiditis is **causally associated** with radiation
             in the **10 to 100** rad dose region.
13
             Plaintiffs have requested that I comment on
14   the defendants['] suggestion that the doubling
             of risk is a necessary piece of evidence in
15   order to establish causation.   I am not an
             attorney and do not know what legal construct
16   may exist regarding this assertion.   I do believe
             it is important for the court to understand that
17   this claim is not well grounded with regard to
             basic principles of epidemiology.   As I have
18   indicated in my previous filings, excess risk
             is but one of the guidelines epidemiologists
19   utilize[] in assessing a **causal relationship.**
             A doubling of the risk is merely one arbitrary
20   point on the continuum of excess risk and as
             such is not required for a finding of **generic**
21   **causation**.

22   The fact that these studies show disease rates that
             are more than twice the background rate seems to
23   be an important consideration in defendants' thinking,
             so I included mention of that fact.   As I said in
24   my report, 'Evidence that the rate of disease in an
             exposed population is higher or that it is
25   significantly higher than the rate for the unexposed
             population may be just as valid as data showing a
26   doubling of the background rate.'

27   (Ruttenber Declaration at pp. 5-6) (Emphasis added).

28   **ORDER RE SUMMARY JUDGMENT-   95**

1   Elsewhere in his declaration, Dr. Ruttenber states: "I do

2 not think a mathematically specified dose-response relationship

3 is always necessary to reach a decision about a **causal**

4 **association**, if there is sufficient alternate evidence." (Id. at

5 pp. 7-8).

6   Dr. Ruttenber's comments pertain to generic causation

7 defined as whether an agent is "capable of causing" a disease.

8 This is the question with which epidemiologists are concerned.

9 As discussed, a "doubling of the risk" is not necessary to prove

10 as an **epidemiological matter** that I-131 is "capable of causing"

11 thyroiditis.[70] It is, however, necessary as a **legal matter** to

12 raise an inference that radiation exposure is a "more likely than

13 not" cause of a disease.

14   Dr. Ruttenber has offered doubling doses for clinical and

15 biochemical hypothyroidism.[71] He has not done so with regard to

16 autoimmune thyroiditis, because he is apparently unable to do so

17 based on the available data.[72] Without evidence of "doubling of

18 _____

     [70] Defendants dispute on other grounds- unscientific
19 methodology- Dr. Ruttenber's conclusion that I-131 exposure is
    "capable of causing" autoimmune thyroiditis. This is discussed
20 infra.

21   [71] Defendants are willing to accept the doubling doses
    Ruttenber offers for non-autoimmune clinical and subclinical
22 (biochemical) hypothyroidism- 750 and 350 rads respectively-
    provided they are increased by a Dose Rate Effectiveness Factor
23 (DREF) of 0.66. DREF is discussed infra in the section
    pertaining to Dr. Radford.
24
     [72] Plaintiffs argue a measurement of "excess relative risk"
25 is not appropriate because it would require an assumption that
    the dose-response risk curve for thyroiditis is linear in the
26 low-dose range and no expert has opined that it is so. They
    argue that a dose-response risk curve for the entire range of
27 doses is not possible because "[i]nsufficient data is available

28 **ORDER RE SUMMARY JUDGMENT-   96**

1 risk," plaintiffs cannot raise an inference that I-131 exposure

2 is a "more likely than not" cause of thyroiditis.

3   The opinion expressed in Dr. Ruttenber's **report** regarding

4 autoimmune thyroiditis does not "fit" the relevant causation

5 inquiry before this court and could not assist a trier of fact in

6 determining whether radiation exposure is a cause in fact of a

7 particular individual's autoimmune thyroiditis.[73]

8

9   **c. Reliability**

10   Because Dr. Ruttenber's opinion is irrelevant, it is

11 technically unnecessary to discuss the scientific reliability of

12 that opinion. However, if there is no admissible evidence that

13 I-131 exposure is even "capable of causing" thyroiditis, then

14 under no circumstances could such exposure be a "more likely than

15 not" cause of thyroiditis.

16   According to defendants, Ruttenber cannot cite one study

17 finding a "causal connection" between **internally** deposited I-131

18 and thyroiditis. Instead, Ruttenber relies on **external** radiation

19 studies (Spitalnik, Nagataki and Kaplan) which, defendants claim,

20 do not satisfy the epidemiological criteria for drawing

_____

to fit such a curve for this disease."
22   This is an admission that a doubling dose for thyroiditis
cannot be derived from the epidemiological data. It is also a
23 peculiar argument by the plaintiffs considering Dr. Mayer used
the same data as Ruttenber (Kaplan and Nagataki) in an attempt to
24 fit a dose-response curve for autoimmune hypothyroidism. It
appears a tacit admission that Mayer did not have sufficient data
25 to fit his curve. Of course, that is exactly what the court has
found to be the case.

26
         [73] This is so whether the causation in fact standard is
27 "but for" or "substantial factor."

28 **ORDER RE SUMMARY JUDGMENT-   97**

1 inferences about a causal association between internally

2 deposited radioiodine and thyroiditis.

3

4   **(1) Spitalnik 1978**[74]

5   In his report, Ruttenber notes that Spitalnik studied

6 thyroid tissue removed from 68 patients given radiation

7 treatments to the head and neck region during childhood for such

8 conditions as enlargement of the thymus, tonsillitis, adenoitis,

9 acne, and scalp problems. They found evidence of chronic

10 lymphocytic thyroiditis "in a large portion of these patients,"

11 and for most patients the doses to the thyroid were less than

12 1,000 rads. Ruttenber observed that because each of the patients

13 had a palpable neck mass, "it [was] difficult to make an estimate

14 of risk per unit dose based on data from this group." (Ruttenber

15 Rpt. at p. 16).

16   According to defendants, Spitalnik does not analyze the

17 doses the patients received, nor does it provide specific dose

18 estimates for any specific conditions under study (including

19 chronic lymphocytic thyroiditis). The authors of the study

20 indicated only that "[i]n most cases, the exact dosage could not

21 be determined, but was less than 1,000 rads." (Spitalnik 1978 at

22 p. 1099). During his deposition, Ruttenber acknowledged

23 Spitalnik did not specify the doses and that it involved "pretty

24 high dose cases." (Ruttenber Dep. at pp. 145-146). The

25 _____

26   [74] Spitalnik et al., "Patterns of Human Thyroid Parenchymal
Reaction Following Low-dose Childhood Irradiation," 41 **Cancer**

27 1098 (March 1978).

28 **ORDER RE SUMMARY JUDGMENT-   98**

1   defendants say that because Spitalnik provides no basis for

2   estimating risk per unit of radiation dose or for evaluating

3   whether there is any dose-response relationship at all, it cannot

4   serve as proof of a cause-effect relationship between I-131 and

5   autoimmune thyroiditis.

6   In his declaration, Ruttenber asserts defendants are wrong

7   in their implication that an "upper limit" on dose- 1,000 rads -

8   is not dose-level information. He claims Spitalnik found an

9   excess disease rate and provided doses "which ranged from zero to

10   one thousand rads."   (Ruttenber Declaration at p. 7).

11   Ruttenber's declaration appears to contradict his deposition

12   testimony that Spitalnik involved "pretty high-dose cases:"

13   Q: Spitalnik & Strauss, you indicate, for
                 most patients the doses to the thyroid
14   were less than 1,000 rad.

15   A: Right.

16   Q: What were the doses?   That's kind of an open-ended
                 phrase.
17
            A: Yeah.   Let's look at them.
18   Well, they don't tell us very much except what I
                 told you.
19
            Q: So it could be 999 rads, you just don't know.
20
            A: Right.   So basically . . . **we would both agree**
21   **that these are pretty high-dose cases.**

22   (Ruttenber Dep. at pp. 145-46)(Emphasis added).[75]

23   Plaintiffs concede they have difficulty getting around this

24   contradiction, asserting that defendants fail to say why the lack

25   _____

26   [75] The court also notes that Ruttenber did not testify 1,000
     rads was an "upper limit," only that most of the exposures were
27   below 1,000 rads.

28   **ORDER RE SUMMARY JUDGMENT-   99**

of a dose-response relationship or a risk estimate "invalidates

the study's use as evidence for a **high-dose effect.**"[76]   In

effect, plaintiffs contend it is methodologically appropriate for

Ruttenber to rely on Spitalnik for the proposition that at **high**

doses, radiation is "capable of causing" autoimmune thyroiditis.

Plaintiffs contend there is no requirement at the "generic

causation phase of the case" to set a specific dose level as

establishing generic causation. Indeed, this is true if "generic

causation" asks only whether the agent is "capable of causing"

the disease. That is a yes or no question which does not

necessarily depend on a dose level.

Plaintiffs concede good information on dose is needed if one

is estimating risk, but they say Dr. Ruttenber was not trying to

make quantitative risk estimates in his report. While that may

be true with regard to his opinion about autoimmune thyroiditis,

it does not square with the specific doubling doses he provides

for non-autoimmune clinical hypothyroidism and biochemical

hypothyroidism.

A dose-response relationship assumes the more intense the

---

[76]   The Kotani rat study (Kotani, et al., "Autoimmune
thyroiditis in the rat induced by thymectomy and low doses of
irradiation: nature of effector cells and demonstration of
antifollicular epithelial cell antibodies," 24 **Clinical
Immunology and Immunopathology**, 111-121) would seemingly also
fall into the "high dose" category. In his report, Ruttenber
cites this study which found that rats treated with thymectomy
and irradiation at doses from 4-8 Gy (**400 to 800 rads**), showed
evidence of autoimmune thyroiditis and proposed a biological
mechanism for the effect.
Neither plaintiffs or defendants discuss Kotani at any
length in connection with the defendants' motion in limine. One
reason may be that it obviously is not a human population study.

ORDER RE SUMMARY JUDGMENT-   100

1 exposure, the greater the risk of disease. The plaintiffs assert

2 there is no requirement for the existence of a dose-response

3 relationship in order to infer causation. Indeed, dose-response

4 relationship is but one of the factors that "guide" an

5 epidemiologist in making a judgment about causation (whether the

6 agent is "capable of causing" the disease). "Reference Guide on

7 Epidemiology," at p. 161. The Reference Guide specifically

8 states that although evidence of a dose-response relationship

9 strengthens the conclusion that the relationship between an agent

10 and disease is causal, it "is not necessary to infer causation."

11 Id. at p. 164.

12   The Reference Guide notes there may not be a dose-response

13 relationship when there is a threshold phenomenon (low dose

14 exposure may not cause disease until the exposure exceeds a

15 certain dose). Id. This is what Ruttenber apparently alluded to

16 at his deposition. Ruttenber acknowledged that biological

17 gradient-of-dose response is important in assessing causal

18 association, particularly with regard to cancer in the low to

19 moderate dose ranges. Ruttenber stated that since thyroiditis is

20 an autoimmune condition and "the biology of autoimmunity is

21 different than the biology of cancer . . . I don't judge this in

22 the same light that I would a cancer dose-response curve in the

23 low dose ranges." (Ruttenber Dep. at pp. 156-57).

24   In other words, Ruttenber says there is a no-threshold

25 phenomenon with regard to cancer (exposure can cause cancer down

26 to the lowest doses) and therefore, one expects a dose-response

27 curve (increasing risk of disease with greater exposure). On the

28 **ORDER RE SUMMARY JUDGMENT-   101**

1   other hand, he suggests there is a threshold phenomenon with

2   regard to autoimmune disease, therefore accounting for a dose-

3   response curve which contains a "plateau" region (i.e. it would

4   not necessarily show increasing risk of autoimmune disease with

5   greater exposure).[77]

6   The fact Spitalnik is a high dose **external radiation** study

7   may limit its **reliability** for establishing that **internally**

8   **deposited** I-131 is "capable of causing" autoimmune thyroid at **low**

9   **dose** levels. However, it cannot be said that Ruttenber's

10   reliance on Spitalnik is unscientific for the most general

11   proposition that radioiodine exposure is "capable of causing"

12   autoimmune thyroiditis.

13

14   **(2) Kaplan 1988**

15   Dr. Mayer relied upon the Kaplan and Nagataki studies in

16   performing a "statistical" analysis which produced a doubling

17   dose for **autoimmune hypothyroidism** at 50 rads. The court deems

18   that analysis scientifically unreliable.

19   Dr. Ruttenber relies upon the same studies in rendering an

20   opinion that I-131 exposure is "capable of causing" **autoimmune**

21   **thyroiditis**. This is a distinct condition, although Ruttenber

22   says it can lead to hypothyroidism. What Dr. Mayer attempts to

23   glean from Kaplan and Nagataki is a significant step beyond what

24   Dr. Ruttenber attempts to glean from the same data. Dr. Mayer

25   _____

26   [77] To the contrary, Mayer's dose-response curve,
     purportedly for autoimmune hypothyroidism, **assumes there is no**
27   **threshold.**

28   **ORDER RE SUMMARY JUDGMENT-**   **102**

produced a dose-response curve and a doubling risk estimate for

autoimmune hypothyroidism. Dr. Ruttenber says he does not have

enough data to fit a curve for **autoimmune thyroiditis** and is not

trying to derive any quantitative risk estimates for that

condition. A quantitative risk estimate is not necessary to

reach a conclusion whether I-131 is "capable of causing"

autoimmune thyroiditis.

Many of the criticisms leveled against Ruttenber for his use

of Kaplan and Nagataki are the same as those leveled against

Mayer. However, just because it is unscientific for Mayer to use

those studies to derive a doubling dose for autoimmune

hypothyroidism (or to opine that low dose exposure is "capable of

causing" autoimmune hypothyroidism) does not necessarily mean

Ruttenber is unscientific in using the same studies to opine very

generally that I-131 is "capable of causing" autoimmune

thyroiditis. In this regard, the court notes also that Ruttenber

is a credentialed epidemiologist and defendants do not take issue

with his knowledge of the radiobiology at issue.

In his report, Ruttenber observes that Kaplan studied 91

women exposed to X-rays during fluoroscopies administered to

evaluate pneumothorax procedures for tuberculosis treatment. The

thyroid doses to the patients ranged between 11-112 rads and

resulted in **"autoimmune thyroid disease"** in 15% of the exposed

and 7% of the control subjects for a prevalence ratio of 2.2 (95%

confidence interval 0.8-6.2). (Ruttenber Report at p. 16).

//

//

ORDER RE SUMMARY JUDGMENT-   103

1 **(a) Specificity**

2   Defendants take issue with Ruttenber's reliance on the

3 Kaplan study because it does not discuss chronic, autoimmune or

4 Hashimoto's thyroiditis, the specific conditions addressed by

5 Ruttenber.   They note the study refers broadly to "autoimmune

6 thyroid disease" which is not specifically defined to include

7 "chronic inflammation" of the thyroid as Ruttenber defines

8 chronic thyroiditis.   (Kaplan 1988 at p. 377).[78] Ruttenber

9 concedes Kaplan's definition includes autoimmune conditions other

10 than thyroiditis and therefore, one cannot derive data from

11 Kaplan pertaining to the specific incidence of chronic

12 thyroiditis.   (Ruttenber Dep. at pp. 151-52).   Thus, say

13 defendants, the "specificity" criterion is not met for the

14 purpose of inferring a causal association between I-131 exposure

15 and chronic thyroiditis.

16   An association exhibits "specificity" if the exposure is

17 associated only with a single disease or **type of disease**.   It is

18 not required that the effect of exposure to an agent be specific

19 for a single disease.   "Reference Guide on Epidemiology," p. 163.

20 According to the Reference Guide, although the presence of

21 "specificity" and "dose-response" strengthens the inference of

22 causation, the absence of either does not weaken the inference.

23 Epidemiologists have begun to question the use of these factors

24 _____

    [78] According to Kaplan, autoimmune thyroid disease was
25 diagnosed when elevated thyroid microsomal anti-body titers were
    found in the presence of at least one of the following: abnormal
26 thyroid palpatory findings, elevated serum TSH, or a history of
    hypothyroidism, hyperthyroidism, or goiter. (Kaplan 1988 at p.
27 377).
28
    ORDER RE SUMMARY JUDGMENT-   104

as guidelines for causation in non-infectious diseases. Id.

In addition to downplaying the significance of the "specificity" criterion, plaintiffs contend that because "autoimmune thyroiditis" is a **type** of autoimmune thyroid disease, there is sufficient "specificity." Plaintiffs note the Kaplan study observed "a higher frequency of autoimmune thyroid disease, either **Hashimoto's thyroiditis** or previously treated Graves' disease in the exposed group." (Kaplan at p. 380). At his deposition, Ruttenber acknowledged Kaplan's "autoimmune thyroid disease" was broad and included autoimmune conditions other than autoimmune thyroiditis, but asserted "they're all autoimmune disease." (Ruttenber Dep. at p. 151). In his declaration, Ruttenber says "specificity" is not a concern with autoimmune thyroid disease because "there is one underlying disease process with a spectrum of severity and few other agents that may confound the relationship between radiation and autoimmune thyroid disease." (Ruttenber Declaration at p. 6).

Dr. Boice, the lead epidemiologist on the Kaplan study, confirms that **Hashimoto's** thyroiditis was included in the category of "autoimmune thyroid disease." Although he says the "broad classification was not ideal," he does not opine that lack of "specificity" makes it impossible for Ruttenber to opine that I-131 is "capable of causing" autoimmune thyroiditis. (Boice Affidavit at Paragraph 26).

The experts can debate the importance of the "specificity" criterion in general, and its significance to Dr. Ruttenber's opinion about the generic causal association between ionizing

ORDER RE SUMMARY JUDGMENT-   105

1 radiation and autoimmune thyroiditis. There is not such a

2 compelling absence of "specificity" that Ruttenber's opinion

3 could be declared unscientific on that basis alone.

4

5   **(b)   Dose Level and Dose-Response**

6   Defendants claim the dose range information in the Kaplan

7 study (11-112 rads) is uncertain because of unaccounted doses

8 from other sources, in particular thyroid scintiscans. They note

9 that Dr. Ruttenber concedes the Kaplan study, like the Spitalnik

10 study, does not provide any information about dose-response

11 relationships. (Ruttenber Dep. at p. 152). Ruttenber

12 acknowledges that from Kaplan he cannot derive a relative risk

13 per unit dose. (Id. at p. 179).

14   Dose-level is not crucial to the essentially yes or no

15 question of whether I-131 is "capable of causing" a disease.

16 Ruttenber confirms this in his declaration where he states that

17 if the true doses for some of the Kaplan study subjects was

18 higher, this would not change his opinion about an association

19 and he would **"simply adjust the dose range where the effect was**

20 **seen."** (Ruttenber Declaration at p. 9)(Emphasis added).

21   The absence of a dose-response relationship is not fatal to

22 inferring whether an agent is "capable of causing" a disease. It

23 is, however, necessary for deriving a relative risk and a

24 doubling dose. In his declaration, Ruttenber states his belief

25 that a "mathematically specified dose-response relationship is

26 [not] always necessary to reach a decision about a causal

27 association, if there is **sufficient alternate evidence."**

28 **ORDER RE SUMMARY JUDGMENT-   106**

1 (Ruttenber Declaration at p. 8). With regard to "alternate

2 evidence, he is referring to consistency of studies, biological

3 plausibility, and timing of the onset of the disease

4 (temporality). These, of course, are the other epidemiological

5 criteria which can be used to support a finding that exposure to

6 an agent is "capable of causing" a particular disease.

7   The uncertainty about dose and the lack of a dose-response

8 relationship in the Kaplan study do not alone render Ruttenber's

9 opinion unreliable. Drs. Boice and Kaplan opine there are

10 limitations to the conclusions which can be drawn from their

11 study. In their respective affidavits, they say it is

12 "inappropriate and unscientific to rely on these data to infer

13 that radiation doses up to 112 rads cause autoimmune thyroid

14 disease or any of the specific conditions included in this broad

15 category." (Kaplan Declaration at Paragraph 16; Boice Affidavit

16 at Paragraph 40). Boice and Kaplan were unwilling to conclude

17 that **their study** established a relationship between autoimmune

18 thyroid disease and **low dose** radiation. (Boice Affidavit at

19 Paragraphs 37-40).

20   At his deposition, Ruttenber acknowledged the Kaplan study

21 authors had nowhere concluded that **low dose** radiation exposure

22 was a risk factor for the development of autoimmune disease.

23 (Ruttenber Dep. at p. 155). However, Ruttenber did not rely on

24 the Kaplan study **alone** in arriving at his opinion that I-131

25 exposure is, in general, "capable of causing" autoimmune

26 thyroiditis. Indeed, in his declaration, Ruttenber claims it is

27 not surprising that Boice and Kaplan were unwilling to reach such

28

ORDER RE SUMMARY JUDGMENT-   107

1 a conclusion since their study was published before the Nagataki

2 and Chernobyl results were published. (Ruttenber Declaration at

3 p. 10). Nagataki is another of the studies upon which Ruttenber

4 relies.[79]

5

6   **(c) Statistical Significance**

7   The Kaplan study reported autoimmune thyroid disease present

8 in 15% of the exposed patients and 7% of the control subjects for

9 a relative risk of 2.2 and a 95% confidence interval of 0.8 and

10 6.2. Ruttenber concedes the Kaplan findings are not

11 statistically significant. (Ruttenber Dep. at p. 148). The

12 range of possible risk (in the confidence interval) includes 1.0

13 which is the normal background incidence of autoimmune thyroid

14 disease.[80] Accordingly, defendants assert this is yet another

15 reason Ruttenber cannot rely on Kaplan for his opinion that I-131

16 is "capable of causing" autoimmune thyroiditis.

17   Relative risk is one of the "cornerstones" for causal

18 inferences.[81] It measures the strength of the association. The

19 _____

20   [79] In his post-report declaration, Ruttenber does not offer
   any epidemiological analysis of the Chernobyl data. He only
   offers the conclusory statement that the data provides further
21 evidence that the autoimmune process can be initiated at doses as
   low as 30 rads in some individuals.

22
   [80] Statistical significance represents the likelihood that
23 the results of an epidemiological study are due entirely to
   chance or random error.

24
   [81] Along with temporality- exposure must occur before
25 development of the disease- there must be some degree of
   statistical association. A weak association may be accepted when
26 demonstrated strength exists in the other epidemiological
   criteria. However, a stronger association provides more evidence
27 for inferring a causal relationship. Thompson, "Causal Inference

28 **ORDER RE SUMMARY JUDGMENT-   108**

1 higher the relative risk, the greater the likelihood the

2 relationship is causal. "Reference Guide on Epidemiology" at p.

3 161. It is important to point out that insofar as determining

4 whether an agent is "capable of causing" a disease (generic

5 causation in the traditional sense), relative risk is but one of

6 the factors (albeit a very important factor) considered by

7 epidemiologists in rendering an opinion about generic causation.

8 In his report, Ruttenber describes "strength of association"

9 between exposure and disease as a measure of the extent to which

10 exposed persons have a disease, as compared with the disease rate

11 in an unexposed population. He observes that strong associations

12 support causality and are less likely to be due to other factors.

13 (Ruttenber Report at p. 4).

14   In his declaration, Ruttenber makes a statement which he did

15 not make in his report and which appears a thinly veiled attempt,

16 based on Kaplan, to rehabilitate his opinion to somehow meet the

17 doubling of the risk, doubling dose criterion for autoimmune

18 thyroiditis. Ruttenber says:

19   I consider the use of Kaplan's odds ratio
            of 2.2 for autoimmune thyroid disease a
20   reasonable value for **autoimmune thyroiditis**.
            For one reason, most of the conditions included
21   in autoimmune thyroid disease will involve
            lymphocytic infiltration and therefore qualify
22   as thyroiditis. Also, based on my review of the
            radiation literature, I do not expect hyperthyroid-
23   ism to substantially alter the finding for auto-
            immune thyroiditis.
24
   (Ruttenber Declaration at p. 10)(Emphasis added).
25

26 _____

    in Epidemiology: Implications For Toxic Tort Litigation," 71
27 **N.C. L. Rev.** 247, 266 and 269 (1992).
28
    ORDER RE SUMMARY JUDGMENT-   109

1   Ruttenber said no such thing in his report and this does not

2   change the fact that the results are not statistically

3   significant. In his report, Ruttenber simply repeated Kaplan's

4   results that there was an odds ratio of 2.2 for **autoimmune**

5   **thyroid disease as a whole.** Ruttenber's declaration is

6   inconsistent with the statement in his deposition that from

7   Kaplan it was impossible to determine the specific incidence of

8   chronic thyroiditis due to the fact Kaplan did not provide such

9   information. (Ruttenber Dep. at p. 152). Kaplan confirms he and

10   Boice did not provide such information:

11   We did not report, and our data do not
             support, a relative risk estimate of 2.2
12   for **autoimmune thyroiditis.** We did not
             separately analyze or present data for
13   autoimmune thyroiditis. . . . the category
             we used - - autoimmune thyroid disease --
14   grouped several different conditions.

15   (Kaplan Declaration at Paragraph 27) (Emphasis added). Kaplan

16   adds it would not be acceptable to substitute the autoimmune

17   thyroid disease category for autoimmune thyroiditis in estimating

18   the dose at which the risk of autoimmune thyroiditis is doubled.

19   (Id. at Paragraph 26).[82]

20   The absence of a statistically significant result in Kaplan

21   indicates at best a weak association between radiation and

22   autoimmune thyroid disease in general, and even more so as to

23   autoimmune thyroiditis specifically. The Kaplan study **by itself**

24   does not support an inference of generic causal association

25   _____

        [82] The Kaplan Declaration and the Boice Affidavit are
26   clearly responsive to assertions made in Ruttenber's declaration
     and therefore, there is no basis for striking them as requested
27   by the plaintiffs.

28   **ORDER RE SUMMARY JUDGMENT-   110**

1 between radiation and autoimmune thyroiditis at **low doses**, in

2 particular the 11 to 112 rads range discussed in Kaplan. Indeed,

3 by itself, Kaplan may not even be sufficient to support the

4 general proposition that radiation is "capable of causing"

5 autoimmune thyroiditis. Ruttenber does not offer an explanation

6 why the lack of a statistically significant result should be

7 ignored in assessing the value of Kaplan.[83]

8

9   **(3) Nagataki 1994**

10   In his report, Ruttenber states Nagataki et al. (1994) found

11 "a significant dose-response relation for persons with antibody

12 positive spontaneous hypothyroidism (or autoimmune thyroiditis)

13 with a maximum odds ratio of about 2.5 at a dose of 0.75 Sv (75

14 rem), and an odds ratio of 2.0 or greater for doses of above 0.4

15 Sv (40 rem)."

16

17   **(a) Specificity**

18   The defendants assert it is unscientific for Ruttenber to

19 _____

20   [83] Statistical significance is not determinative of
    causation. It merely reflects the likelihood that study results
21 are due to chance or random error. Because statistical
    significance depends on study size, a small study could yield an
22 unbiased, correct result and yet suffer from lack of statistical
    significance. Thompson, "Causal Inference in Epidemiology:
23 Implications For Toxic Tort Litigation," 71 **N.C. L. Rev.** 247, 257
    (1992).
24   At his deposition, Ruttenber stated in regard to Kaplan that
    because it dealt with "small numbers" and there is a point
25 estimate greater than 1 (within the confidence interval of 0.8 to
    6.2), if the numbers are increased up to a point of adequate
26 statistical power, that "point estimate becomes significant."
    However, Ruttenber also acknowledged that it could "go in the
27 other direction." (Ruttenber Dep. at pp. 148-49).

28
    ORDER RE SUMMARY JUDGMENT-   111

1 place any reliance on Nagataki as support for an association

2 between external radiation and thyroiditis (chronic, autoimmune,

3 and Hashimoto's) because Nagataki expressly disclaims these

4 disease classifications. In other words, defendants say the use

5 of Nagataki flunks the "specificity" criterion. According to

6 Nagataki:

> Hashimoto's disease or autoimmune thyroiditis
> was not included in the classification of
> thyroid disease in the present study because
> the definition of the disease differs among
> experts, except for the histological findings,
> which were difficult to obtain from all subjects
> in the present study. However, we used the
> criterion of autoimmune hypothyroidism, in which
> patients have increased serum TSH levels with
> or without decreased serum thyroid hormone levels
> (clinical or subclinical hypothyroidism) and
> positive thyroid autoantibodies, because there
> is much less disagreement regarding this criterion.

14 (Nagataki at p. 366).

15   When confronted with this passage during his deposition,

16 Ruttenber conceded Nagataki was not dealing with the specific

17 conditions of Hashimoto's disease and autoimmune thyroiditis, but

18 was dealing with a "precursor" to those conditions. According to

19 Ruttenber, " . . . Nagataki supports the mechanism of

20 autoimmunity . . . ." Ruttenber conceded Nagataki does not

21 provide information as to the extent to which the autoimmune

22 process results in **clinical** disease. In turn, he also conceded

23 Nagataki does not provide information from which a determination

24 can be made as to the "association of radiation dose and a

25 **clinical** condition" including Hashimoto's disease and autoimmune

26 thyroiditis. Ruttenber testified that Nagataki presents evidence

27 "for the early stages in terms of the proposed mechanism of the

28 **ORDER RE SUMMARY JUDGMENT-   112**

1   disease."   (Ruttenber Dep. at pp. 163-64).

2   Plaintiffs note the Nagataki study explicitly states that

3   its results "suggest . . . the prevalence of antibody-positive

4   spontaneous hypothyroidism (**autoimmune thyroiditis**) is

5   increased."   (Nagataki at p. 364)(Emphasis added).   According to

6   plaintiffs, the defendants have confused autoimmune thyroiditis

7   which has been determined "biochemically," from autoimmune

8   thyroiditis which has been determined by diagnosis of obvious

9   symptoms (**clinically**).   The plaintiffs cite references which they

10   claim support the notion that biochemical autoimmune thyroiditis

11   is the same condition as antibody-positive spontaneous

12   hypothyroidism.   Thus, contend plaintiffs, this explains why

13   Nagataki can say the "the prevalence of antibody-positive

14   spontaneous hypothyroidism (autoimmune thyroiditis) is increased"

15   and simultaneously say "Hashimoto's disease or autoimmune

16   thyroiditis was not included in the classification of thyroid

17   disease in the present study."   Plaintiffs assert that because

18   Nagataki used a biochemical definition of autoimmune thyroiditis

19   means some persons may have the disease without having obvious

20   symptoms.   Although this may raise an issue about compensability

21   of the disease as a "physical" injury[84], plaintiffs contend it

22   does not raise a <u>Daubert</u> issue.

23   Ruttenber's declaration is in accord with this explanation,

24   asserting Nagataki studied autoimmune thyroiditis that was

25   determined biochemically, and not from symptoms found by an

26   _____

27   [84] See discussion <u>infra</u>.

28   **ORDER RE SUMMARY JUDGMENT-   113**

1 examining physician. He provides two theories why Nagataki

2 states that Hashimoto's disease and autoimmune thyroiditis were

3 not included in the classification of thyroid disease: 1)

4 Nagataki could have actually removed cases showing overt symptoms

5 of Hashimoto's disease which explains the dip in the Nagataki

6 curve at the highest level; 2) Nagataki used a biochemical

7 diagnostic technique that included classical overt cases, as well

8 as those more difficult to diagnose.

9   The defendants contend these are just speculative theories

10 and Ruttenber does not know which one is correct. According to

11 defendants, it shows that Ruttenber cited Nagataki without taking

12 this issue into account. Defendants also note that in his

13 declaration, Ruttenber acknowledges the possibility of a

14 misclassification bias arising from a biochemical definition of

15 autoimmune thyroiditis which includes clinical cases as well as

16 subclinical ones. (Ruttenber Declaration at p. 8). Defendants

17 observe that Ruttenber's report says nothing about a

18 misclassification bias.

19   In assessing the reliability (methodological soundness) of

20 Ruttenber's opinion, it is important to consider the scope of

21 that opinion: ionizing radiation is "capable of causing"

22 autoimmune thyroiditis. Frankly, the court does not find

23 anything in Ruttenber's report and deposition which is glaringly

24 inconsistent with what he says about Nagataki in his subsequent

25 declaration. In his deposition, there is the following exchange

26 between counsel and Ruttenber:

27   Q: Doctor, I look at these studies, and I see

28 **ORDER RE SUMMARY JUDGMENT-   114**

1   that they are addressing different things, and
     that's what I'm trying to discuss with you.

2

3   A: See, I don't think they are addressing
     different things. I think that there is a
     spectrum of effect from abnormal antibodies

4   to evidence of tissue effect induced by
     autoimmunity. So I think they [the studies]

5   are all providing different pieces. They are
     looking at different endpoints, perhaps.

6

7   . . . I agree that there is not a wealth of
     literature on this, but what I've seen puts
     together a **reasonable picture for the ability**

8   **of radiation to cause autoimmune disease. You**
     know, the problem is there has not been a

9   **lot of work.**

10   (Ruttenber Dep. at pp. 167-68)(Emphasis added).

11   Nagataki is one of the "pieces" upon which Dr. Ruttenber

12   relies. Although Nagataki alone may not be helpful in

13   determining the association of radiation dose and **clinical**

14   Hashimoto's disease and autoimmune thyroiditis, the court is not

15   convinced it has absolutely no value for that proposition when

16   viewed along with other studies (Spitalnik and Kaplan). If the

17   question is the association between ionizing radiation and

18   biochemical autoimmune thyroiditis, Nagataki alone may suffice.

19   Ruttenber's use of Nagataki cannot be discredited based on

20   the "specificity" criterion, especially when the "Reference Guide

21   on Epidemiology" indicates this criterion is satisfied if the

22   exposure is associated with a type of disease (in this case,

23   autoimmune disease) and further indicates that the absence of

24   "specificity" does not weaken the inference of causation

25   (assuming, of course, the other criteria are satisfied).

26   //

27   //

28   **ORDER RE SUMMARY JUDGMENT-   115**

(b) Dose-Response

Defendants contend Ruttenber's "unqualified" assertion that Nagataki's curve shows a maximum odds ratio of about 2.5 at a dose of 0.75 Sv (75 rem) is "unsupported." Defendants note that Nagataki's curve peaks at 70 rads, falls to less than 2.0 at doses of 100 rads and less than 1.0 at doses of 120 rads. It is a "concave" curve. As dose increases, the risk does not continue to increase, but eventually decreases.

At his deposition, Ruttenber readily acknowledged the curve is "concave."[85] According to Ruttenber:

> . . . my point is that [Nagataki] found a dose-response -- one, that he found a doubling of effect of doses in his ranges. But what he did -- it is true that the higher dose ranges, the risk came back down.

(Ruttenber Dep. at p. 156). As noted above, Ruttenber explains that biological gradient-of-dose response (increasing risk with increasing dose) is found with regard to cancer in the low to moderate dose ranges, but he asserts the "biology of autoimmunity" is different from the "biology of cancer" and therefore, can be attributable for the variation in the dose-response curve for antibody-positive spontaneous hypothyroidism (autoimmune thyroiditis). (Ruttenber Dep. at p. 157). According to Ruttenber, autoimmune disease can produce "strange" dose-response curves because much depends on whether individuals are at "high risk genotype for sensitivization" and so some people with very low exposures may get the disease, while some people

---

[85] Dr. Mayer, however, insists the curve is "convex."

ORDER RE SUMMARY JUDGMENT-   116

1 with very high exposures may not get the disease. (Ruttenber

2 Dep. at p. 159).

3   Defendants point out that Ruttenber was unable to explain

4 the inconsistency between Nagataki finding a decreased risk at

5 doses above 100 rads and the findings of the Spitalnik study

6 showing increased risk at high doses (about 1,000 rads).

7 Ruttenber acknowledged there was an inconsistency in the data

8 which needed to be explained and that he did not try to explain

9 it in his report. (Ruttenber Dep. at p. 161).

10   If Ruttenber was opining there is an association between

11 ionizing radiation and autoimmune thyroiditis at **low doses**, this

12 "inconsistency" might make such an opinion scientifically

13 unreliable. However, it does not necessarily render unreliable

14 Ruttenber's fundamental premise that there is an association

15 between ionizing radiation and autoimmune thyroiditis at **some**

16 **unspecified dose level.** In his **report**, Ruttenber was not

17 specific about a dose-range, a threshold dose or a doubling dose.

18 It may be precisely because of "inconsistency" in the data that

19 Ruttenber felt he could not be dose-specific. Therefore, he

20 merely recited the results of the studies without extrapolating

21 to either a dose range, a threshold dose or a doubling dose.

22   It is true Nagataki opines that because his dose-response

23 curve is concave, there is a need for further studies "on

24 relatively **low dose** radiation effects on thyroid disease."

25 (Nagataki at p. 370). However, Ruttenber does not specifically

26 assert in his **report** that there is a generic causal association

27 between **low doses** of ionizing radiation and autoimmune

28 **ORDER RE SUMMARY JUDGMENT-   117**

1   thyroiditis. He concludes only that there is a causal

2   association between ionizing radiation and autoimmune

3   thyroiditis. At his deposition, Ruttenber was careful to say

4   that Nagataki, "coupled with . . . other evidence (i.e. high dose

5   study like Spitalnik) . . . suggests that there is an effective

6   (sic) radiation in the causing of autoimmune disease." Ruttenber

7   says that Nagataki is "a piece of evidence that shows that there

8   is a relation between radiation and autoimmune disease."

9   (Ruttenber Dep. at pp. 157-58).[86]

10   The opinion expressed in Ruttenber's report, **and the only**

11   **one that counts**, is that ionizing radiation is "capable of

12   causing" autoimmune thyroiditis- not that ionizing radiation is

13   "capable of causing" the condition specifically at low doses.

14   Fed. R. Civ. P. 26(a)(2)(B) provides that the expert report is to

15   contain a **complete** statement of all opinions to be expressed and

16   the basis and reasons therefor, and the data or other information

17   considered by the witness in forming the opinions. Accordingly,

18   the court will not consider the assertion in Ruttenber's

19   declaration that Kaplan and Nagataki provide evidence of a causal

20   association in the 10 to 100 rad dose range.

21   The "Reference Guide on Epidemiology" indicates the absence

22   of a "linear" dose-response relationship (the more intense the

23   exposure, the greater the risk of disease) is not necessary to

24   infer generic causation. Therefore, the existence of a "concave"

25   ─────────────────

26   [86] It is not intrinsically "unscientific" for experienced
     professionals to arrive at a conclusion by weighing all available
     scientific evidence. General Electric Company v. Joiner, 118

27   S.Ct. 512, 522 (1997) (Stevens, concurring).

28   **ORDER RE SUMMARY JUDGMENT-   118**

1 dose response curve in Nagataki is not sufficient to find

2 Ruttenber's reliance thereon scientifically improper for his

3 **limited opinion that ionizing radiation is "capable of causing"**

4 **autoimmune thyroiditis.** If Ruttenber now wishes to opine that

5 ionizing radiation is "capable of causing" autoimmune thyroiditis

6 at low doses, that is a different proposition and one that may

7 well not be methodologically sound based on the Spitalnik, Kaplan

8 and Nagataki studies as a whole. This is due in particular to

9 the limitations of Kaplan and Nagataki cited by defendants- i.e.

10 lack of statistical significance and unaccounted for doses in the

11 Kaplan study; concave dose-response curve in Nagataki.

12

13   **(c) Consistency**

14   Defendants assert Nagataki does not satisfy the

15 "consistency" criterion. They note Ruttenber's deposition

16 testimony that Nagataki is the first study that analyzed a dose-

17 response relationship for autoimmune thyroid disease. (Ruttenber

18 Dep. at p. 160). Therefore, say defendants, Ruttenber "had no

19 basis for comparing the concave dose-response" observed by

20 Nagataki.

21   Defendants observe that a number of other epidemiological

22 studies have investigated the relationship between radiation and

23 thyroiditis and have found no association. Defendants fault

24 Ruttenber for not citing these studies which dealt specifically

25

26

27

28 **ORDER RE SUMMARY JUDGMENT-   119**

1 with thyroiditis: Yoshimoto 1995[87], Morimoto 1987[88] and Kerber

2 1993.[89]

3   Plaintiffs argue the finding of inconsistent results in

4 different populations does not nullify the results of one study

5 for "general causation," unless the study results are likely to

6 have occurred by chance. Plaintiffs assert that all one has to

7 show in "general causation" is the disease is causally associated

8 with radiation in at least one population. In other words,

9 plaintiffs contend the fact Yoshimoto, Morimoto and Kerber

10 reached different results concerns the "weight" of Dr.

11 Ruttenber's opinion and not its admissibility. According to

12 plaintiffs, Dr. Ruttenber's opinion is admissible so long as it

13 is supported by other studies such as Spitalnik, Kaplan and

14 Nagataki. The court agrees.

15   Research findings are often replicated in different

16 populations. Consistency in these findings is an "extremely

17 important factor" in making judgments about causation. Different

18 studies which examine the same exposure-disease relationship

19 should yield similar results. Any inconsistencies signal a need

20 _____

21   [87] Yoshimoto, et al., "Prevalence Rate of Thyroid Diseases
Among Autopsy Cases of the Atomic Bomb Survivors in Hiroshima,
22 1951-1985," **Radiation Research** (1995).

23   [88] Morimoto, et al., "Serum TSH, Thyroglobulin, and
Thyroidal Disorders in Atomic Bomb Survivors Exposed in Youth:
24 30-Year Follow-up Study," 28 **Journal of Nuclear Medicine** 1115
(July 1987). Plaintiffs' Ex. 100 to Appendix 4B re Iodine
25 Claims.

26   [89] Kerber, et al., "A Cohort Study of Thyroid Disease in
Relation to Fallout From Nuclear Weapons Testing," 27 **Journal of
27 the American Medical Association** (Nov. 1993).

28 **ORDER RE SUMMARY JUDGMENT-   120**

1   to question whether the relationship is causal. "Reference Guide

2   on Epidemiology" at p. 162.

3   Ruttenber offers a limited, narrow opinion that I-131 is

4   "capable of causing" autoimmune thyroiditis. Ruttenber says

5   nothing about dose and nothing about risk. Viewed in that

6   context, it is not obvious that Ruttenber's opinion flunks the

7   "consistency" criterion such that it can categorically be

8   declared scientifically unreliable.

9   Defendants' argument that there is an inconsistency between

10   Kaplan and Nagataki due to differences in their respective

11   definitions of autoimmune thyroid disease go to the "weight" of

12   Ruttenber's opinion. The same is true with regard to their

13   arguments about other studies showing no causal association

14   between radiation exposure and thyroiditis.[90]

15

16   **d. Conclusion**

17   "Epidemiology has its limits at the point where an inference

18   is made that the relationship between an agent and a disease is

19   causal (general causation) **and where the magnitude of excess risk**

20   **attributed to the agent has been determined; that is,**

21   **epidemiology addresses whether an agent <u>can cause a disease</u>, not**

22   **whether an agent <u>did cause a plaintiff's disease</u>.** "Reference

23   Guide on Epidemiology," at p. 167 (Emphasis added).

24   _____

25   [90] If Ruttenber's opinion was that there is a causal
     association at low doses (below 100 rads), then the consistency
     criterion is not met because Nagataki is all there is to support
26   that proposition. Kaplan is no support for **low dose** causal
     association due to the lack of statistical significance and the
27   failure to take into account additional thyroid doses.

28

ORDER RE SUMMARY JUDGMENT-   121

1   Dr. Ruttenber, a "credentialed epidemiologist," limited

2 himself to the question of whether I-131 "can cause" autoimmune

3 thyroiditis. In arriving at his opinion that I-131 "can cause"

4 autoimmune thyroiditis, he relied on several of the guidelines

5 which epidemiologists consider in making a judgment about

6 "generic causal association." There is no requirement that each

7 factor of Hill's or Koch's postulates be satisfied to infer

8 "generic causal association."[91] The absence of supporting

9 factors, such as "specificity" and "dose-response relationship"

10 do not render Ruttenber's methodology scientifically unreliable,

11 although those considerations affect the "weight" which should be

12 afforded his opinion.

13   For the limited, general proposition that ionizing radiation

14 is "capable of causing" autoimmune thyroiditis **at some**

15 **unspecified dose**, Ruttenber's opinion (**as set forth in his**

16 **report**) cannot be stricken for lack of reliability. However,

17 that is all which can be derived from his **report**. That is not

18 the same as an opinion that radioiodine is "capable of causing"

19 autoimmune thyroiditis at **low doses**. The court will not consider

20 the opinion found in Ruttenber's post-report and post-deposition

21 declaration that Kaplan and Nagataki provide evidence that

22 autoimmune thyroiditis is causally associated with radiation in

23 the 10 to 100 rad dose range.

24   The opinion found in Ruttenber's report is also insufficient

25

26   [91] Although as mentioned above, at a minimum, "temporality"
must be satisfied and there must be some level of statistical
27 association.

28

ORDER RE SUMMARY JUDGMENT—   122

1 to warrant a jury's consideration of any plaintiff's thyroiditis

2 claim.   The court will therefore exclude that opinion based on

3 the fit/relevancy prong (Prong 2) of Daubert.

4

5 **3. Edward Radford**

6 **a.   Introduction**

7 Dr. Radford is an epidemiologist and a medical doctor.   He

8 submitted a November 14, 1995 iodine report on behalf of the

9 plaintiffs entitled "Comments on the Medical Findings Associated

10 With Exposure to Radioactivity From the Hanford Facility in

11 Washington."   It was supplemented in March 1996.

12 Dr. Radford served on the prestigious BEIR I and BEIR III

13 Committees (Biological Effects of Ionizing Radiation).   He

14 chaired the BEIR III Committee.   The defendants contend that

15 although Radford was once a credentialed epidemiologist, he is no

16 longer so.   They note he did not serve on the BEIR IV or BEIR V

17 Committees.   Defendants point out that Radford's proffered expert

18 testimony has been stricken in a number of previous cases as

19 being scientifically unreliable.

20 Although Dr. Radford's committee and publishing activity may

21 have fallen off in recent years, his resume leaves no doubt he is

22 qualified to render epidemiological opinions about the

23 relationship between I-131 and neoplastic and non-neoplastic

24 thyroid conditions.   The fact his expert opinions have been

25 stricken in previous cases cannot excuse the court from a

26 thorough, objective analysis of the particular opinions he offers

27 **in this case.**   The defendants do not maintain that the opinions

28 **ORDER RE SUMMARY JUDGMENT-   123**

1 offered by Dr. Radford in the instant case are the same as those

2 opinions stricken in previous cases.

3

4   b. **Neoplastic Thyroid Conditions (Thyroid Cancer)**

5   Dr. Radford offers what he believes are the doses at which

6 the risk of contracting thyroid cancer is doubled by exposure to

7 I-131. According to Radford:

8   To determine the dose ranges . . . I have
           assumed that an excess relative risk of 100%
9   is sufficient to establish causality of
           thyroid cancer by radiation. I have used a 100%
10   increase in relative risk, a so-called doubling
           of the relative risk, as a basis for estimating
11   causative dose ranges in order to be conservative.

12 (Radford 1995 Iodine Report at p. 25).

13   The doubling doses ultimately arrived at by Radford are as

14 follows: 1) individuals ages 0-4: 1 rad; 2) individuals ages 5-

15 9: 2 rads; 3) individuals ages 10-19: 7 rads; and 4) individuals

16 ages 20 and older: 20 rads.

17

18   (1) **Dose Rate Effectiveness Factor (DREF)**

19   In computing his doubling doses, Radford did not take into

20 account a "dose rate of effectiveness factor" (DREF). A DREF

21 assumes internally deposited I-131 is not as effective as

22 **external radiation** in inducing cancer and biological damage. The

23 defendants contend it is scientifically improper for Radford to

24 not include a DREF since his doubling doses are derived from a

25 study of a population exposed to high dose rate **external**

26 **radiation**, that being the atomic bomb survivors. Thompson, et

27 al., "Cancer Incidence in Atomic Bomb Survivors. Part 2: Solid
28
    ORDER RE SUMMARY JUDGMENT-   124

Tumors, 1958-1987," 137 **Radiation Research** S17 (1994).[92] If a

DREF were applied to Radford's doubling doses, it would **increase**

those doses. The defendants say Radford should have used the

0.66 DREF recommended by the BEIR V committee. An 0.66 DREF

means I-131 is only 66 percent as effective as external radiation

in inducing thyroid cancer.

Radford offers the following rationale for not employing a

DREF:

> One issue that has been discussed in the literature
> has been the relative effectiveness of radioactive
> iodine exposure in producing thyroid cancer, compared
> to exposure to external x-rays or gamma rays. From
> the mass of evidence now available in human
> populations, as well as recent animal studies (Laird,
> 1987), it appears that any differences between these
> two modes of irradiation are not great, and the
> general consensus is that I-131 is very similar in
> its carcinogenic potential as an equal dose from
> external gamma or x-radiation (BEIR V, p. 294, 1990,
> Laird 1987). Moreover, the fact that people downwind
> of the Nevada test site also developed an excess of
> thyroid abnormalities including cancer, from radiation
> exposures to I-131 over several years, supports the
> view that thyroid cancer effects of exposure can occur
> when doses are delivered at low dose rates (Kerber,
> et al., 1993), compared to high dose rates in the
> A-bomb study. The exposure conditions in Utah are
> comparable to those at Hanford.

(Radford 1995 Iodine Report at pp. 17-18).

The defendants claim Radford's description of the evidence

is misleading in that rather than there being a "mass of human

data," there is no human data establishing I-131 and external

radiation are equally effective, nor is there a "general

consensus" that the relative effectiveness of the two is similar.

----

[92] Plaintiffs' Ex. 126 to Appendix 4C re Iodine Claims;
Plaintiffs' Ex. 47 to Appendix 4 re Non-Iodine Claims.

ORDER RE SUMMARY JUDGMENT-   125

The defendants offer an impressive list of organizations, in addition to BEIR V (1990)[93], which have endorsed a reduction factor: UNSCEAR (United Nations Scientific Committee on the Effects of Atomic Radiation) 1994[94] (UNSCEAR 1994 at p. 4) ("Iodine 131 **appears** to be less effective than external radiation in causing thyroid cancer, **perhaps** by a factor of 3-5"); NCRP (National Committee on Radiation Protection and Measurements) 1985[95], 1993[96] (NCRP 1985 at p. 31) ("Iodine-131 **appears** less carcinogenic in people on a rad-for-rad basis than external radiation; how much less is yet to be determined . . . [but] an upper limit value of one-third is recommended for application to the general population, **until additional data becomes available**"); National Academy of Sciences Committee Reviewing the Arctic Aeromedical Laboratory's Thyroid Function Study (NRC 1996[97] at p. 31) ("Iodine-131 is **estimated** to be 20-25 percent as effective as externally administered x-rays in producing thyroid cancer . . . . the reason for this low carcinogenic potential is not **well understood** . . . .").

---

[93] NRC, **Health Effects of Exposure to Low Levels of Ionizing Radiation** BEIR V (1990). Defendants' Ex. 6.

[94] UNSCEAR, **Sources and Effects of Ionizing Radiation: UNSCEAR 1994 Report to the General Assembly,** (1994).

[95] NCRP, "Induction of Thyroid Cancer by Ionizing Radiation," (**NCRP Report 80**)(March 30, 1985). Defendants' Ex. 88.

[96] NCRP, "Risk Estimates for Radiation Protection," (**NCRP Report 115**)(Dec. 31, 1993). Defendants' Ex. 89.

[97] NRC, "The Arctic Aeromedical Laboratory's Thyroid Function Study: A Radiological Risk and Ethical Analysis" (1996).

ORDER RE SUMMARY JUDGMENT-   126

1   Defendants also point out that plaintiffs' experts, Dr. A.

2   James Ruttenber and Dr. Kelly Clifton, have endorsed the BEIR V

3   estimate of an 0.66 DREF. In his 1995 iodine report, Ruttenber

4   says he endorses using the BEIR V estimate for carcinogenesis and

5   that it is "reasonable to use this estimate for endpoints other

6   than cancer **until contrary evidence is available."** (Ruttenber

7   1995 Iodine Rpt. at p. 12; Dep. at pp. 80-81). In his

8   deposition, Clifton stated that taking into account both human

9   and animal data, I-131 **"may be as much as 66 percent"** effective

10   as external radiation. Clifton added he thought the figure was

11   around there, although he was not happy with any of the human

12   data. (Clifton Dep. at p. 90).

13   The obvious point to be derived from all of this is that .66

14   is not an absolute figure. It is a figure which represents the

15   scientific judgment of the BEIR V Committee. That of course,

16   does not mean it is methodologically sound for Dr. Radford not to

17   employ any DREF at all. However, it does mean there is room for

18   a difference of opinion, provided a scientifically reliable basis

19   for a different opinion is articulated.

20   The plaintiffs claim defendants are challenging the "weight"

21   of Dr. Radford's opinion about the DREF, rather than the Daubert

22   admissibility of that opinion. The defendants say that is not

23   so. The problem, according to the defendants, is that Radford

24   snatched his opinion out of thin air without knowing the

25   scientific basis for it. Defendants say Radford came up with his

26   opinion first and looked to support it only when the opinion was

27   challenged. This, say defendants, reflects an unscientific

28   **ORDER RE SUMMARY JUDGMENT-   127**

1 methodology.

2   At his deposition, Radford acknowledged BEIR V's "best

3 estimate" was 66 percent, but asserted that "they [BEIR V] could

4 not rule out an equal sensitivity." (Radford Dep. at p. 324)

5 According to Radford, BEIR V could not distinguish the

6 comparative effectiveness factor from one ("1") (equal

7 effectiveness). (Id. at 331). Indeed, according to the BEIR V

8 report:

9   The risk ratio estimate so derived for [I-131]
            compared to x-rays was 0.66 (95% confidence limits,
10   0.14-3.15) and did not differ significantly from 1.0.
            [Laird 1987].
11

12 (BEIR V at p. 294). The confidence interval includes 1.0.

13   Dr. Laird utilized both animal and human data. She

14 concluded the data provided no compelling evidence to suggest the

15 risks accompanying external radiation or I-131 exposure were any

16 different. (Laird 1987[98] at p. 1). However, she also concluded

17 that using both types of data, a 66% effectiveness rate factor

18 was appropriate. This figure (66%) was a compromise between the

19 human studies which suggested considerably lower potencies for I-

20 131 relative to external radiation, and animal studies which

21 showed a slightly higher potency for I-131. (Laird at p. 306).

22   Defendants seize upon the following passage from Radford's

23 deposition testimony as indicating he did not understand Dr.

24 Laird's analysis, nor the basis of the BEIR V endorsement of a

25 0.66 effectiveness factor:

26   [98] Laird, et al., "Thyroid Cancer Risk from Exposure to
Ionizing Radiation: A Case Study in the Comparative Potency
27 Model," 7 **Risk Analysis** 299 (1987). Defendants' Ex. 73.

28 **ORDER RE SUMMARY JUDGMENT-   128**

1 Q: What was the Laird paper based on Doctor?

2 A: Based on a review of the situation as he saw it.

3 Q: First of all, Doctor Laird is a she, correct?

4 A: She, yes, sorry, excuse me.

5 Q: What was she analyzing in her paper?

6 A: I can't tell you right now.

7 Q: All you know about Laird is that it had to do
   with animals?

8

   A: No, it also reviewed the human data.

9

   Q: And what did she conclude with respect to
10   the human data analyzed alone?

11 A: I can't recall specifically, but the . . .
       BEIR V committee concluded that the figure
12   of 0.66 was appropriate, and they cited Laird.

13 Q: And isn't the 0.66 figure based on combining
       the animal data and the human data?
14

   A: Oh, I don't know. I'm not sure what basis they
15   have.

16 Q: And you don't know how the 0.66 was derived?

17 A: Not exactly, no.

18 (Radford Dep. at pp. 332-333). Defendants also point out that

19 Dr. Radford did not know the official position of the NCRP and

20 other such organizations as to the dose rate effectiveness

21 factor. (Id. at 333-34).

22   Certainly, it is not enough for Radford just to say that

23 because Dr. Laird and BEIR V could not rule out equal

24 effectiveness, equal effectiveness is a scientifically reliable

25 theory. He has to state **why** it is a scientifically reliable

26 theory. Despite their concession to the possibility of equal

27 effectiveness, Laird and BEIR V concluded a .66 dose rate

28 **ORDER RE SUMMARY JUDGMENT-   129**

effectiveness factor was appropriate. All Dr. Radford's original

1995 iodine report provides is a conclusion about equal dose rate

effectiveness without offering any scientific reasoning for

distinguishing Laird's analysis that .66 is an appropriate dose

rate effectiveness factor.

In March 1996, Dr. Radford submitted a supplemental iodine

report which revisited the issue of equal dose effectiveness.

This time, he discussed some of the deficiencies in the human

data used to conclude that internally deposited I-131 is not as

effective as external radiation in causing biological damage.

The human data was derived from studies of therapeutic and

diagnostic uses of low doses of I-131 (Hoffman 1994; Holm, et al.

1988; and Hall, et al. 1996). The patients given the doses

included very few persons under age 20. However, a very

substantial fraction of all the excess cases of thyroid cancer

for a general population appear in those irradiated at young

ages- below 20- as confirmed by the Thompson A-Bomb study

(Thompson, et al. 1994). According to Radford, the absence of

individuals under age 20 makes the human data misleading for

inferring I-131 is not as effective as external radiation.

(Radford Supp. Rpt. at pp. 1-2).

In his supplemental report, Radford also refers to Chernobyl

data which he says supports his opinion of equal effectiveness.

According to Radford, "[t]he new information from the Chernobyl

studies strongly indicates that [I-131] with its 8 days half-life

is at least as effective in producing thyroid cancer in children

as external radiation." (Id. at p. 2). At his deposition,

ORDER RE SUMMARY JUDGMENT-   130

1 Radford elaborated upon this, pointing to the findings of

2 plaintiffs' expert, Dr. Viktor Ivanov. Radford testified it was

3 possible to derive approximate risk co-efficients from the Ivanov

4 results which "appear to agree quite well with the A-bomb

5 results." (Radford Dep. at p. 327). "Primarily" because of

6 this, Radford said he was of the opinion I-131 is as effective as

7 external radiation. (Id. at 332).

8   As defendants point out, neither Radford's 1995 iodine

9 report or his 1996 supplemental report mentions Ivanov. The

10 first reference to Ivanov occurred at Radford's deposition.

11 According to defendants, during Radford's deposition testimony it

12 was apparent he did not know what was actually shown by Ivanov's

13 analysis. Defendants cite the following deposition testimony:

>   Q: Did the [Ivanov] report . . . show a
>   dose-response relationship?
>
>   A: They said that in the range of, I think
>   it was 10 to 60 rads, there was more
>   than a two-fold increase in the thyroid
>   cancers compared to background or normal
>   rate. That, to me, is a kind of dose
>   response curve right there.
>
>   Q: What happened to the higher dose
>   categories and the lower dose categories?
>
>   A: I don't know. We didn't mention those.
>
>   Q: So in your analysis of a dose-response
>   relationship, you didn't look to see
>   what the trend was among the different
>   dose categories for which he presented
>   data?
>
>   A: He didn't present data for these different
>   dose categories.

26 (Radford Dep. at p. 335).

27   Defendants note that for his **5-60 rad** dose category, Ivanov

28
ORDER RE SUMMARY JUDGMENT-   **131**

showed a relative risk of 0.46 (negative risk) and confidence

intervals of 0 and 2.1 (not statistically significant). (Ivanov

Dep. at 84-85). Thus, say defendants, Radford quoted only the

upper bound of the confidence interval (2.1) and ignored

everything else in asserting the category showed a two-fold

increase in risk. Defendants also note that Ivanov did have

"different" dose categories: 60-140 rads and "greater than 140

rads." Defendants concede the 60-140 rads category showed a

statistically significant excess, but claim that because the

analysis was based on a small sample, it lacks statistical power

and the confidence intervals are broad, ranging from 1.8 to

38.9.[99] According to defendants, Radford conceded during his

deposition that Ivanov's data was not statistically robust and

contained wide confidence intervals:

> Q: Let me show you Figure 1 in Dr. [Ivanov's]
>   report. Do you regard that chart as
>   reflecting statistically robust data?
>
> A: Not as it is presently presented no. But
>   this became the case control study.
>
> Q: How wide are the confidence intervals?
>
> A: They are pretty wide.

(Radford Dep. at p. 337).

In response, plaintiffs say that at his deposition, Dr.

Radford "mistakenly" referred to Ivanov's 5-60 dose category. In

his declaration (Ex. 7 to Plaintiffs' Appendix 1 re Iodine

Claims), Radford acknowledges the mistake:

---

[99]   Ivanov's analysis is also the subject of a motion in
limine which is discussed infra.

ORDER RE SUMMARY JUDGMENT-   132

> I mistakenly took the low dose end of his two lowest dose ranges, 5 to 60 rad and 60 to 140 rad as the average dose applicable. . . . There is still, nevertheless, a radiation dose-response relationship for excess thyroid cancer demonstrated by his data.

What Radford apparently is referring to is Ivanov's 60-140 dose category which shows a relative risk of **7.15** per Sievert with confidence intervals at 1.8 to 38.9 for children 0-18 years of age. (Figure 5 at p. 11 of Ivanov 1995 Iodine Report). According to plaintiffs, this compares well with the **7.3** relative risk per Sievert estimate for children 0-19 "derived from the Thompson A-bomb data cited in Dr. Radford's report" and therefore, confirms his opinion about the similarity between the Chernobyl data and the A-bomb data and the equal effectiveness between I-131 (Chernobyl) and external radiation (A-bomb).

Defendants say plaintiffs' reference to "raw" Chernobyl data is unavailing because the data is "unpublished, unreviewed and unreliable as a basis for a bona fide epidemiological opinion and . . . whatever support [they] may eventually lend to Radford, if any, they do not change the unscientific methodology he used to generate his original opinion." Defendants add that the fact plaintiff's expert Dr. Baruch Modan may agree with Radford's conclusion (Modan Dep. at 174) says nothing about Radford's methodology. Defendants reassert that because Radford coughed up his equal effectiveness opinion first and looked for support only when he was challenged on it, his methodology is unscientific and his opinion inadmissible per Daubert.

If all Radford was going on was his original 1995 report,

ORDER RE SUMMARY JUDGMENT-   133

1 his opinion regarding equal effectiveness would require exclusion

2 because it is conclusory. However, in his supplemental report,

3 Radford mentions limitations in the I-131 human data (includes

4 very few children). Furthermore, although he does not mention

5 Dr. Ivanov's analysis in his supplemental report, Radford

6 discusses therein the "new information from the Chernobyl

7 studies" and asserts it strongly indicates I-131 is at least as

8 effective in producing thyroid cancer in children as external

9 radiation.

10 When asked at his deposition if Ivanov's analysis was

11 contained in any of his reports, Radford acknowledged it was not,

12 but that he did not know of Ivanov's involvement in the case

13 until "comparatively recently." (Ivanov Dep. at p. 327). That

14 is arguably not very compelling, considering Ivanov's iodine

15 report (containing his analysis of the Chernobyl data) is dated

16 October 1995, before both Radford's original iodine report and

17 his supplemental report. And certainly, Radford did not give a

18 stellar performance at his deposition in explaining how Ivanov's

19 analysis bolstered his (Radford's) "equal effectiveness" opinion.

20 On the other hand, what the court may perceive as a lack of

21 sufficient information and articulation in Radford's original and

22 supplemental reports about the evidence supporting equal

23 effectiveness (limitations in the human data; the Chernobyl

24 data), may simply not be true for the reason that his reports are

25 addressed to the scientific community which is already aware of

26 the assumptions underlying his opinion. Defendants have not

27 offered an affidavit from any of their experts challenging Dr.

28 ORDER RE SUMMARY JUDGMENT-   134

1   Radford's opinion about equal effectiveness, challenging the

2   assertion that human I-131 data has limitations because it

3   includes few children, or challenging reliance upon Chernobyl

4   data.

5   Defendants say the scientific evidence at best establishes a

6   doubling dose for thyroid cancer at 50 rads for individuals

7   between ages 10-19 at the time of exposure, and 16 rads for

8   individuals between ages 0-9 at the time of exposure. This is

9   based on figures from plaintiffs' expert Dr. Kelly Clifton which

10   take into account a DREF of 0.66. At his deposition, Clifton

11   stated he thought the DREF was "around there," (0.66), but added

12   "I can't tell you, because I'm not happy with any of the human

13   data." He also testified that what he found overwhelming and

14   important about the Chernobyl data was the total number of

15   thyroid cancers that occurred there, suggesting equal potency of

16   I-131 was not beyond the pale. (Clifton Dep. at pp. 90-91).

17   Considering the equivocal status of the 0.66 DREF opined by

18   BEIR V, Dr. Clifton, Dr. Ruttenber, Dr. Modan and others, the

19   court will allow Dr. Radford to testify about equal effectiveness

20   (and the bases thereof). If necessary, a jury can assess the

21   weight of Radford's opinion versus that of BEIR V. The equivocal

22   status of the 0.66 DREF overrides any shortcomings in Radford's

23   analysis, shortcomings which defendants will probably use in an

24   attempt to impeach Radford before a jury.

25   Defendants' motion in limine will be denied with regard to

26   Radford's opinion about DREF.

27
28
     ORDER RE SUMMARY JUDGMENT-   135

(2) Risk Estimates

(a)   Age Adjustments

The Thompson A-bomb study reports a relative risk for children ages 0-9 of 9.5 per Sievert. In his 1995 report, Radford said that to be "conservative" in assessing the risk of thyroid cancer after irradiation from I-131, he would adopt a risk coefficient for radiation induction of thyroid cancer under the age 10 as 10% per rem, which is "close" to the risk estimate derived from Thompson. (Radford 1995 Iodine Rpt. at p. 22). Furthermore, Radford opined:

> In order to take account of the greater risk
> for those irradiated from 0-4 years of age
> (Ron, Modan, et al., 1989), which showed that
> the risk for children under age 5 was about
> twice that for children aged 5-9, a figure of
> 20% per rem for this 0-4 age group is appropriate,
> as well as those exposed in utero to [I-131] through
> their mothers during pregnancy.

(Id.) The result is to decrease the doubling dose in half for individuals ages 0-4 versus those ages 5-9, as reflected in Radford's final double dose figures: 1) individuals ages 0-4: 1 rad; 2) individuals ages 5-9: 2 rads.

Defendants assert that in failing to make a "corresponding adjustment" to the 5-9 age group, the effect is a "double counting" and an overstating of the risk for the 0-4 age group. According to defendants, a boost in the risk estimate for the 0-4 age group should result in a corresponding decrease in the risk estimate for the 5-9 age group. However, Radford leaves the 5-9 age group with the 10% per rem risk estimate he derived from the Thompson data. Defendants explain it as follows:

ORDER RE SUMMARY JUDGMENT-   136

If, as Thompson suggests, the risk for the **entire**
0-9 age group is 9.46% per rem, then 9.46% must
be the **average** risk for Radford's 0-4 and 5-9
age groups. Under the Radford regime, however,
the average risk would be **15%** ((20% + 10%) [/] 2
= 15%). Thus, by increasing the risk co-efficient
for the 0 to 4 age group to 20% per rem while leaving
the risk co-efficient for the 5-9 age group at 10%
per rem, Radford has **double counted** and inflated the
risk for this group as a whole by a substantial margin.

(Defendants' emphasis).

During Radford's deposition, the following exchange

occurred:

> Q: But Doctor, if you don't make an adjustment
>   for the 5 to 9 group, while you are increasing
>   the 0 to 4 group, aren't you double counting?
>
> A: Well, not necessarily. As I say, it depends
>   on the proportions of children in the various
>   age groups that were present in the study
>   population.
>
> Q: Have you done any analysis to insure that you
>   are not double counting?
>
> A: **No, I haven't.**

(Radford Dep. at p. 341)(Emphasis added).

In his 1995 report, Radford indicated the need for a higher

risk estimate for the 0-4 age group came from the Ron, Modan, et

al., 1989 tinea capitis study involving patients in Israel

irradiated for ringworm of the scalp[100]:

> The Israel study of over 10,000 children
> involved those children irradiated at an
> average age of 7 years (1-15 years). In
> this group the excess thyroid cancer relative
> risk per sievert is 32.5, that is, 32.5% per
> rem. In the case of the A-bomb survivors
> (Thompson, _et al._, 1994), the excess relative

---

[100]   Ron, et al., "Thyroid neoplasia following low-dose
radiation in childhood," 120 **Radiation Research** 516-531 (1989).
Plaintiffs' Ex. 108 to Appendix 4B re Iodine Claims.

ORDER RE SUMMARY JUDGMENT-   137

risk for children aged 0-9 is 9.5 per sievert,
that is, 9.5% per rem, and for children aged
10-19 is 3.0 per sievert, that is 3.0% per rem.
It is apparent that there is a divergence in the
results of the investigation of these two study
populations, since, for the youngest age groups
in the tinea capitis study, the dose required to
double the risk of thyroid cancer is about 3 rems,
whereas for the A-bomb survivors it is 10.5 rems
. . . .

The Israeli study (Ron, Modan, et al., 1989)
looked also at a comparison of thyroid cancer
risks of those who were exposed under the age
of 5 with those children exposed aged five and
over. This comparison showed that the excess
relative risk for children under the age of five
was about twice as great for those exposed
aged five to nine. Such a finding is consistent
with the pronounced effect of age at exposure found
for the A-bomb survivors as well as other irradiated
groups (Ron, Modan, et al., 1989). Those irradiated by
radioiodine in utero (Johnson, 1982) may be at similar
high risk.

(Radford 1995 Iodine Rpt. at pp. 21-22).

Defendants assert that during his deposition, Radford

changed his reliance from the tinea capitis study (Ron, Modan, et

al., 1989) to a pooled analysis of external radiation studies by

Ron (E. Ron et al., "Thyroid Cancer After Exposure to External

Radiation: A Pooled Analysis of Seven Studies," 141 **Radiation**

Research 259 (1995)).[101] Defendants note that Radford had the

Ron 1995 pooled data available to him during preparation of his

1995 report, but say he missed its presumably critical point

about doubled risk among children aged 0-4. According to

defendants, while Ron supports the concept of a greater risk for

0-4 year olds, it provides no basis whatsoever for the risk

estimates employed by Radford. Defendants assert that neither

---

[101]   Defendants' Ex. 106.

ORDER RE SUMMARY JUDGMENT-   138

Thompson or Ron support the risk estimates Radford proposes for

the 0-4 and 5-9 age groups and therefore, Radford has no basis

whatsoever for his "risk-hiking testimony."

In his 1995 report, Radford referred to the pooled Ron

analysis (Ron 1995) as summarily evaluating seven independent

studies, including the Thompson A-bomb study and the 1989 tinea

capitis study. (Radford 1995 Iodine Rpt. at p. 21). At his

deposition, Radford discussed how he believed the Ron 1995 pooled

data supported his risk co-efficients for the age groups 0-4 and

5-9:

> Q: Now the source of your 10 percent
>   per rem is what?
>
> A: Well, it is very close to the figure
>   given by Thompson. Now, what I did
>   was look at the data in the Ron report
>   [Ron 1995] and . . . for less than age 15
>   they reported 7.7 as the figure which
>   would be the excess relative risk per
>   sievert. . . . I consider 10 below the
>   age of 10 and 7.7 below the age of 15
>   to be virtually identical because of the
>   rapid fall-off in risk after the age 10.

(Radford Dep. at p. 340).

When asked whether he was basing his adjustments to the

lower age groups on the 1989 tinea capitis data, Radford

responded "[n]o, I am basing it on both data. . . . [i]t has been

done for the A-bomb data, for example, in Ron [Ron 1995]."

Radford testified that Ron 1995 found an effect "very similar to

the Tinea Capitis study." (Radford Dep. at pp. 342-43). Later,

during Radford's deposition, plaintiffs' counsel registered an

objection and asserted Radford had not based his risk estimates

on the tinea capitis study. (Radford Dep. at p. 346). Radford

ORDER RE SUMMARY JUDGMENT-   139

1   responded that "initially" he had based his risk estimates on the

2   tinea capitis study until he found out what Ron 1995 had done to

3   the A-bomb data: "pull out the 0-4 group, and they came out with

4   a two-fold increased risk comparing the 5 to 9 with the 1 to 4

5   (sic) group." (Id. at pp. 346-47).

6   At his deposition, Radford acknowledged Ron 1995 was

7   available to him at the time he prepared his 1995 report.

8   Radford admitted that at the time he prepared the report, he had

9   "missed this point" about the Ron 1995 pooled data. According to

10 Radford, during the preparation of his original report, he did

11 his "best" to read Ron 1995: "I read it quickly. I absorbed the

12 general point, and that's what I was armed with at the time."

13 After that, Radford says he began to look at the report [Ron

14 1995] "carefully . . . and noticed this graph." (Radford Dep. at

15 pp. 347-48). Radford acknowledged Ron 1995 does not provide risk

16 estimates for the 0-4 and 5-9 age groups:

17   They [Ron 1995] didn't pull out individual age
            groups beyond the issue of less than 15. That's
18   all they did. They didn't say anything about
            1 to 4, except to say that it was much higher
19   than 5 to 9 and much higher than- greater than
            10 to 14, I think it was.

20
            So that's all they said on this. They did not
21   calculate an excess risk per sievert for that
            particular age group, and they did it for their
22   own reasons. I don't know why, but the mere fact
            that they don't say it in here doesn't mean that
23   you can't infer it from the data.

24 (Radford Dep. at pp. 350-51).

25   Plaintiffs offer no reason why Radford missed the

26 significance of the Ron 1995 pooled data when he prepared his

27 report. Radford apparently did not discover this in time to

28
ORDER RE SUMMARY JUDGMENT-   140

1   incorporate it in his March 1996 supplemental report.

2   It is important that Radford never disclaimed the validity

3   of the analysis contained in his 1995 report which is based on

4   the A-bomb study and the 1989 tinea capitis study. Radford

5   essentially states the Ron 1995 pooled analysis confirmed the

6   effect seen from the 1989 tinea capitis study. The court

7   disagrees with defendants that Radford unequivocally withdrew

8   reliance upon the 1989 tinea capitis study.[102] Although the

9   defendants claim any increase in the risk estimate for the 0 to 4

10 age group requires a corresponding downward adjustment in the

11 risk estimate for the 5 to 9 age group, they do not take issue

12 with the notion that an increase is appropriate for the 0 to 4

13 age group. What defendants contend is that neither Thompson or

14 Ron provide any basis for the actual risk estimates sought to be

15 used by Radford (20 rem for children 0 to 4 and 10 rem for

16 children 5 to 9).

17   On the other hand, plaintiffs contend it is scientifically

18 appropriate for Radford to draw the inferences which he has drawn

19 from the data. According to plaintiffs, defendants never state

20 or explain how or why Radford's interpretation is illogical,

21 unreasonable or unscientific based on the data reported.

22 Plaintiffs say Radford was conservative in starting out with a

23 10% per rem figure for children under age 10 based on the A-bomb

24 data, as compared to the 32.5% per rem figure reported in the

25 _____

26   [102] This is so even though **plaintiffs' counsel** stated during
    the deposition that Radford's risk estimates were not based upon
27 the 1989 tinea capitis study.

28 **ORDER RE SUMMARY JUDGMENT-   141**

1   1989 tinea capitis study. Based on this divergence, they assert

2   it is reasonable for Radford to double the 10% per rem figure for

3   the 0-4 age group, while leaving the 5-9 age group at the 10% per

4   rem figure. At his deposition, Radford stated his use of 10 for

5   the 5-9 group was probably low based on the Ron 1995 pooled

6   analysis. (Radford Dep. at p. 344). As plaintiffs observe, the

7   defendants do not seem to challenge Radford on this particular

8   point.

9   In his declaration, Radford addresses the defendants'

10   concern about "double counting" in his splitting of the 0-4 and

11   5-9 age groups. Radford states he did not make a "mathematical

12   error," as claimed by the defendants, because he did not employ a

13   mathematical approach:

> When I considered the younger children, I
> referred to the Israeli study by Modan, Ron
> and others cited in my reports, since that
> was the only study where the ages at exposure
> were divided into two age groups, 0 to 4 and
> 5 to 9. The risk coefficients for thyroid
> cancer from the Israeli study were substantially
> higher than for the Japanese A-bomb results,
> and I stated in my report that I was conservative
> (i.e. probably underestimating the risk) in
> using the Japanese coefficients. When I came to
> estimate the risk for children aged 0 to 4, I
> chose to consider **all the evidence available**, and
> on this basis I chose the value I did.

(Radford Declaration, Ex. 7 to Plaintiffs' Appendix 1 re Iodine

Claims at Paragraph 7) (Emphasis added).[103]

_____

[103] According to Justice Stevens' concurring opinion in the
recent decision of General Electric Company v. Joiner, 118 S.Ct.
512, 523 (1997), "[i]t is not intrinsically 'unscientific' for
experienced professionals to arrive at a conclusion by weighing
all available scientific evidence-- this is not the sort of
'junk' science with which Daubert was concerned."
    Also, see the underlying Court of Appeals opinion which

ORDER RE SUMMARY JUDGMENT-   142

1   Defendants' position on Radford's age adjustments is too

2   extreme.   Just because Radford's figures are not reported in

3   Thompson or Ron does not mean his analysis is "unscientific."

4   Inferences can be derived from existing data provided the

5   inference is derived by the scientific method. Daubert II, 43

6   F.3d at 1316 (9th Cir. 1995). Here again, as with Radford's DREF

7   opinion, the defendants have not offered an opinion from any of

8   their experts challenging either Radford's methodology or his

9   "inference" on age adjustments as drawn from the Thompson and Ron

10   data.   It cannot be said that Radford's opinion amounts to no

11   more than "subjective belief or unsupported speculation."

12   Hopkins v. Dow Corning Corp., 33 F.3d 1116, 1124 (9th Cir. 1994),

13   citing Daubert I, 113 S. Ct. at 2795.

14   Radford has been in the radiation health effects field for

15   quite some time. At least with regard to DREF and age

16   adjustments, he is testifying about matters that grow naturally

17   and directly out of research he has conducted independent of this

18   litigation.   Defendants do not assert his opinion has been

19   developed for the express purpose of offering testimony in this

20   _____

21   stated:

22   Opinions of any kind are derived from individual
            pieces of evidence, each of which itself might
23   not be conclusive, but when viewed in their
            entirety are the building blocks of a perfectly
24   reasonable conclusion, one reliable enough to
            be submitted to a jury along with the tests
25   and criticisms cross-examination and contrary
            evidence would supply.

26   78 F.3d 524, 532 (11th Cir. 1996).

27
28   **ORDER RE SUMMARY JUDGMENT-**   143

1 case.   Accordingly, the fact Radford has yet to publish his

2 opinion on DREF and age adjustments in the peer-reviewed

3 literature is not significant.   Defendants do not assert

4 otherwise.

5   As with Radford's opinion about DREF, the issue concerning

6 Radford's age adjustments is really the persuasiveness or

7 correctness of his opinion.   That, of course, is not for the

8 court to analyze as part of its gatekeeper function.   That is an

9 issue of "weight" for the trier of fact.

10   Defendants' motion in limine will be denied as regards

11 Radford's age adjustments for children below age 10.

12

13   **(b)   Individual Susceptibility Factor**

14   Radford's doubling doses for thyroid cancer incorporate an

15 individual susceptibility factor of five.

16   Assuming the equal effectiveness of I-131 and external

17 radiation, and the necessity for an upward adjustment in the 0 to

18 4 age group, Radford's doubling doses for thyroid cancer are: 1)

19 for individuals ages 0-4 at the time of exposure: 5 rads; 2) for

20 individuals ages 5-9 at the time of exposure: 10 rads; 3) for

21 individuals ages 10-19 at the time of exposure: 33 rads; and 4)

22 for individuals ages 20 and above at the time of exposure: 100

23 rads.

24   With his individual susceptibility factor, Radford further

25 reduces each of these doubling doses: 1) individuals ages 0-4:

26 1 rad (5 rads divided by factor of 5); 2) individuals ages 5-9:

27 2 rads (10 rads divided by factor of 5); 3) individuals ages 10-

28 **ORDER RE SUMMARY JUDGMENT-   144**

19: 6.66 rads (rounded to 7 rads) (33 rads divided by factor of

5); and 4) individuals ages 20 and above: 20 (100 rads divided

by factor of 5).

Radford contends the effect of individual sensitivity,

"particularly for thyroid cancers and adenomas" must be taken

into account:

> The subject of individual susceptibility has
> recently been addressed by a committee of the
> U.S. National Academy of Sciences (Isselbacher,
> et al., pp. 200-03, 1994). Its conclusions are:
> for about 1% of the population, human cancer
> susceptibility to the environmental agents can
> vary by a factor as large as 100-fold. For
> about 5% of the population the variation could
> be 25-fold. Application of such large factors
> at this time, however, pending further confirmation,
> does not appear to me to be appropriate. Again to
> continue in a conservative position, I have taken
> a range of special susceptibility from **genetic**
> and other medical conditions to be a factor of
> five (Finkel, 1995). On this basis individual
> excess relative risk coefficients for the four age
> groups [0-4; 5-9; 10-19; above 20] **may** range over
> at least a factor of five above that reported in
> the heterogeneous populations studied
> epidemiologically.

(Radford 1995 Iodine Rpt. at pp. 23-24) (Emphasis added).

It is not clear if Radford ever identifies the specific

medical conditions which affect susceptibility. It appears his

emphasis is on genetic susceptibility:

> [I]nitiation and promotion of cancer, including
> thyroid cancer, is a multi-stage process, and
> several environmental factors may interact to lead
> finally to a cancer developing. Nevertheless,
> exposure to radiation **may** be a major factor resulting
> in the development of cancer, particularly thyroid
> cancer. Even though there is reason to believe
> that contributing agents to cancer induction are
> widespread, the epidemiology shows that radiation
> is the major cause. For this reason, the contribution
> to causation required for radiation to be a major
> contributing factor to the development of cancer

ORDER RE SUMMARY JUDGMENT-   145

1   would be less than a 100% increase in relative risk,
      as determined from epidemiologic results. If we
2   assume the development of a human cancer is like
      filling a glass, which when filled leads to a growing
3   cancer, there may be several factors which go to
      fill the glass. Examples are background radiation,
4   inhalation or ingestion of irritant chemical agents,
      or virus infections that can disrupt normal cell
5   structures. Thus, exposure to the initiating effect
      of ionizing radiation will not be the only factor
6   filling the glass and leading to cancer. But if
      exposure is sufficient, it can be considered the major
7   contributing cause of the cancer, particularly for
      thyroid cancer for which radiation is the only **known**
8   cause. 'Sufficient' does not mean radiation is the
      only contributing cause. **The effect of the concept**
9   **of genetic susceptibility to cancer discussed above**
      is that the glass for such individuals is smaller,
10   **and thus more easily filled.**

11   (Radford 1995 Iodine Rpt. at pp. 25-26)(Emphasis added).

12   It is important to emphasize that the issue is individual

13   susceptibility to **radiation-induced** cancer. After all, what we

14   are attempting to determine is whether Hanford's radiation

15   emissions are a "more likely than not" cause of thyroid cancer.

16   Cancer, in general, has a number of different causes. And even

17   for thyroid cancer, for which Radford asserts radiation is the

18   only **known** cause, the fact is there may be unknown causes.

19   Furthermore, individuals may have been exposed to sources of

20   radiation other than Hanford emissions. Once again, the point is

21   that it is necessary to distinguish causes to some reasonable

22   extent in order to fairly assign culpability to Hanford

23   emissions.

24   Defendants contend Radford's individual susceptibility

25   factor is an arbitrary litigation-driven proposal which is not

26   the product of scientific analysis or investigation. They say

27   his proposal finds no support in either the scientific literature

28
     ORDER RE SUMMARY JUDGMENT-   **146**

1   cited by him (Isselbacher 1994[104] and Finkel 1995[105]) or among

2   scientists in the field.   Furthermore, defendants assert Radford

3   does not and cannot specify any currently available method for

4   identifying plaintiffs who are more susceptible to contracting

5   radiation-induced cancer, nor can he quantify individual

6   variations in susceptibility.

7   According to defendants, neither the Isselbacher or the

8   Finkel article recommends or supports the five-fold increase in

9   risk co-efficients proposed by Radford.   Defendants claim these

10   articles are "thought-pieces" which merely discuss the concept of

11   individual susceptibility, but do not recommend that objectively

12   derived radiation risk-estimates be multiplied.   Defendants note

13   the Isselbacher article concerns carcinogenic risk associated

14   with exposure to hazardous air pollutants, not radiation.   They

15   further point out that the Isselbacher article concludes "[t]he

16   population distribution of interindividual variation in cancer

17   susceptibility cannot now be estimated with much confidence."

18   (Isselbacher 1994 at p. 207).

19   According to defendants, Finkel acknowledges this as well,

20   stating "the correct functional form of this distribution [the

21   extent of susceptibility across the population] is not known."

22   (Finkel at p. 304).   Furthermore, defendants point out the

23   _____

         [104]   Isselbacher, et al., **Science and Judgment in Risk
24   Assessment (Committee on Risk Assessment of Hazardous Air
     Pollutants, NRC)**, (1994).   Defendants' Ex. 54.

25
         [105]   Finkel, **A Quantitative Estimate of the Variations in
26   Human Susceptibility to Cancer and Its Implications for Risk
     Management, in Low Dose Extrapolation of Cancer Risks: Issues
27   Papers**, (Stephen Olin et al., eds)(1995).

28   **ORDER RE SUMMARY JUDGMENT-   147**

following quotes from Finkel as evidencing how little is known

about individual susceptibility and the factors involved:

> [C]omparatively little evidence exists to validate
> the theory that inherent differences among people
> contribute substantially to the observed variation
> in adverse outcomes. . . . [W]e do not yet know how
> much of this enormous difference in apparent
> susceptibility is due to specific characteristics
> of those involved and how much is due to the
> stochastic [random] nature of the carcinogenic process.

(Finkel at p. 299).

> Many of the most important biologic determinants
> of susceptibility are probably 'hidden' in the
> sense that science cannot at present readily
> discern who the most susceptible and who the
> most resistant persons are . . . .

(Finkel at p. 320).

Plaintiffs argue Isselbacher and Finkel support the

proposition that there is variation in susceptibility among

populations, even if it is not subject to estimation or

quantification. Plaintiffs contend the defendants are

essentially criticizing Radford for "extrapolating reasonably"

from Isselbacher and Finkel. They argue there is no requirement

in science or law that evidence used in support of a position

must come from a source "in which the authors take precisely the

same position, to exactly [the] same degree of specificity."

According to plaintiffs, if Radford failed to compensate for

susceptibility in light of Isselbacher and Finkel, "he would have

seriously underestimated the risks to those among the population

who are more susceptible than the median person for which the

population risk numbers are derived." Thus, he introduced his

"conservative factor of five."

ORDER RE SUMMARY JUDGMENT-   148

Radford's susceptibility adjustment only increases his cancer risk estimates and that he has not proposed any downward adjustment for those who are less susceptible. Defendants argue this omission is particularly glaring due to the fact both Isselbacher and Finkel indicate that if some percentage of the population is more susceptible to cancer, a corresponding percentage would be less susceptible. Indeed, Isselbacher states:

> The study [Finkel 1987] concluded that as a first approximation, the amount of variability (for either sex, either disease, and either country) could be roughly modeled by a lognormal distribution with a logarithmic standard deviation on the order of 2.0 . . . . That is, about 5% of the population might be about 25 times more susceptible than the average person (and a corresponding 5% about 25 times **less susceptible**); about 2.5% might be 50 times more (**or less**) susceptible than the average, and about 1% might be at least 100 times more (**or less**) susceptible.

(Isselbacher 1994 at p. 203) (Emphasis added).

At his deposition, Radford was asked whether he proposed to adjust risk estimates to reflect the lower susceptibility of certain members of the population. Radford responded in the negative, asserting "the lower susceptibility is the normal susceptibility who have no genetic abnormalities." Radford denied that there were people who were "[m]uch less likely to get cancer than normal people." Radford stated he had not seen any reference in the scientific literature to such an idea. (Radford Dep. at p. 288-89).

When confronted with the Isselbacher language, Radford was forced to change his position:

ORDER RE SUMMARY JUDGMENT-   149

1   Q: Do you disagree with this Isselbacher?

2   A: No, I don't disagree with Isselbacher. I
         think the point they make, that about
3   5 percent of the population might be
         25 times more susceptible than the average
4   person is a reasonable conclusion.

5   Q: Do you disagree with Isselbacher when he
         says that 5 percent could be 25 times less
6   susceptible?

7   A: **I am disagreeing only with the concept that
         the log normal mathematical analysis demands
8   this, but what it doesn't say is how much—
         oh, yes, it does say. It says about 25 times
9   less susceptible. I don't quite know what
         that means. Some people will never get cancer.**
10
     Q: As presented, do you disagree with it?
11
     A: I am only saying that the idea that there could
12   be a tail of individuals with high risk is clear.
         That's consistent with Isselbacher. The question
13   of whether there is an equally long tail that is
         . . . 25 times . . . less susceptible, I'm not
14   sure about that.

15   Q: **Have you _ever_ analyzed the range of susceptibility
         within a population?**
16
     A: **No.**
17
     Q: If you were to determine that susceptibility does
18   follow a log normal distribution within a
         population, would you be prepared to adjust your
19   risk estimates to account for those people who are
         less susceptible?
20
     A: Yes, I think so.
21
     Q: Are you aware of any tools that are available today
22   to identify those persons who are less susceptible
         to cancer?
23
     A: No.
24
(Radford Dep. at pp. 298-99) (Emphasis added).
25
         This deposition testimony does not reflect favorably upon
26
Radford's individual susceptibility factor which concerns itself
27
28
ORDER RE SUMMARY JUDGMENT-   150

1 only with "greater" susceptibility, and ignores the possibility

2 of "lesser" susceptibility. It shows Radford has no basis for

3 his increased susceptibility factor. Especially telling is

4 Radford's admission that he has never analyzed the range of

5 susceptibility within a population.   Isselbacher and Finkel

6 simply do not provide the necessary scientific support.

7   It is appropriate to contrast Radford's DREF and age

8 adjustment opinions which do find sufficient support within the

9 scientific community to at least warrant presentation to a jury.

10 With regard to the DREF, even BEIR V acknowledged the possibility

11 of equal effectiveness between I-131 and external radiation, plus

12 Radford cited other arguably legitimate grounds for equal

13 effectiveness. At his deposition, Radford conceded no scientific

14 organization had endorsed his approach of increasing risk

15 estimates to account for increased susceptibility. He indicated

16 that such organizations were just beginning to look at the

17 concept. (Radford Dep. at pp. 299-300).

18   With regard to age adjustments, the defendants do not

19 dispute that the 1989 tinea capitis study and the Ron 1995 pooled

20 data support an increased risk estimate for the 0 to 4 age

21 category. Radford's figures- 20% per rem for the 0 to 4 group

22 and 10% per rem for the 5 to 9 group- are not necessarily beyond

23 what the scientific literature will support. On the other hand,

24 Isselbacher and Finkel do not support an increased risk estimate

25 for increased susceptibility. Unlike the case with his equal

26 effectiveness opinion and his age adjustments, Radford has gone

27 too far from what the scientific literature will reasonably

28 **ORDER RE SUMMARY JUDGMENT-   151**

1 support insofar as individual susceptibility.[106]

2   The defendants argue, and reasonably so, that since the

3 underlying epidemiological studies on which baseline cancer risk

4 estimates are based include persons of **all** levels of

5 susceptibility, the risk co-efficients derived from the studies

6 incorporate the entire range of susceptibility within the

7 population, and there is no need for adjustments to account for

8 individual susceptibility. Say defendants:

> If Radford wants to adjust these baseline risk
> estimates for persons of **greater** susceptibility,
> then he must first **remove** them from the underlying
> epidemiological studies, because their inclusion
> skews the risk estimates **upwards** due to their
> greater susceptibility. Once the hypersensitive
> individuals are factored out of the underlying
> studies, the risk estimates for the remaining
> population should **drop**, and the doses necessary
> to prove generic causation should **rise**. Radford's
> proposed decrease in the doubling dose would then
> apply to doubling doses that are **substantially larger
> than those** currently in use which represent the **entire
> range of individual susceptibilities**.

(Defendants' Br. at 40)(Emphasis in text).

      The plaintiffs' response is that it is precisely because the

risk estimates derived from the population studies incorporate

the "entire range of individual susceptibilities," that it is

necessary to single out those with greater susceptibility to

radiation-induced cancer because they will experience a doubling

dose at a much lower exposure. Say plaintiffs:

> The underlying premise of [defendants'] argument,
> of course, is that the risk co-efficients are

---

[106] On that basis, the absence of testimony from a
defendant's expert specifically refuting Radford's individual
susceptibility factor is not a compelling reason to give Radford
the benefit of the doubt.

ORDER RE SUMMARY JUDGMENT-   152

> **averages** and **therefore apply to the fictional average person.** The population surrounding the facility, however, was not a homogenous population of hundreds of thousands of varying individuals.

(Plaintiffs' Response Br. at p. 41).

The court is not persuaded by plaintiffs' argument. The risk co-efficients are indeed averages, but the fact is those risk estimates are derived from **heterogenous** populations, just like the Hanford population, which incorporate the whole range of susceptibilities. (See Radford 1995 Iodine Rpt. at p. 24- "[I]ndividual excess relative risk coefficients for the four age groups may range over at least a factor of five above that reported in the **heterogenous** populations studied epidemiologically"). Those risk estimates take into account age and sex which, according to Radford, are the **major** modifiers of risk. (Radford Dep. at p. 295).

Plaintiffs assert they are not deriving risk estimates for the "average person" living around Hanford, but rather for a select group of individuals: those who have lived around the Hanford facility, who were exposed to releases from the Hanford facility, and who present with a "radiation related" disease. According to plaintiffs:

> [They] are a group of people who have a higher-than-average susceptibility to radiation induced cancer, or another way of saying it is that the plaintiffs are a group of people whose cells are more likely to be damaged, and not quickly repaired by radiation than a randomly selected group of people from the same population.

(Plaintiffs' Response Br. at p. 44). Plaintiffs say this is so based on "standard statistical theory."

ORDER RE SUMMARY JUDGMENT–   153

1   In other words, plaintiffs contend their greater than

2 average susceptibility to radiation induced cancer is already

3 established by the fact they have a "radiation related" disease.

4 This is too great a leap. It requires an assumption that the

5 plaintiffs' cancers were in fact radiation-induced. That cannot

6 be assumed merely on the basis of living near Hanford and being

7 exposed to some level of Hanford emissions. Radiation has not

8 been isolated as the exclusive cause of thyroid cancer. Radford

9 asserts radiation is the only "known" cause, but that leaves

10 unknown causes.[107]

11   In his deposition, Radford acknowledged there are "many"

12 causes of cancer and that there are individuals who are more

13 susceptible to cancer "caused by a variety of agents." He

14 acknowledged the difficulty of identifying individuals who are

15 susceptible to radiation-induced cancer as opposed to cancer

16 caused by another agent "because they may be susceptible **not only**

17 **to radiation, but to other agents.**" He acknowledged that he did

18 not have a tool available for identifying those persons more

19 susceptible to radiation-induced cancer, but not to cancers from

20 other agents. He acknowledged that at present it was not "easy"

21 to define a group of people who are more susceptible to cancer

22 from all causes (not just radiation-induced cancer). (Radford

23 Dep. at pp. 302-305) (Emphasis added).

24   Defendants cite deposition testimony from Drs. Modan and

25 ───────────────

26   [107] Even if a greater than average susceptibility to
   radiation could be established, that of course would still leave
   the question of whether Hanford radiation could be tagged as the
27 source of the cancer versus some non-Hanford source of radiation.

28 **ORDER RE SUMMARY JUDGMENT-   154**

Clifton which they say confirms persons who are more susceptible

to radiation-induced cancer cannot be identified, nor can

differences in susceptibility be quantified. Plaintiffs assert

that while these experts stated they could not quantify

variability, they did not say such a procedure was unscientific

or impossible. Nonetheless, the court finds nothing from these

other experts amounting to an endorsement of Radford's

application of an individual susceptibility factor to objectively

derived risk estimates.

Referencing Radford's deposition testimony (pp. 289-90),

plaintiffs assert that among the means of distinguishing between

sensitive and non-sensitive persons is the use of family

histories and differential diagnosis. According to plaintiffs,

if a "particular plaintiff" identifies a family or individual

history of thyroid disease or cancer, or identifies a family or

individual history of sensitivity to radiation, "it is more

likely this person will also be in the increased group of

sensitivity."

One of the problems with this is exactly how an individual

is supposed to identify a family or individual history of

sensitivity specifically to radiation. Radford says there

currently is no tool available to do this, such as a laboratory

analysis which allows discernment of the genetic make-up of

individual cells. (Radford Dep. at p. 290). A family history of

thyroid cancer or disease is not by itself going to establish

increased sensitivity to radiation-induced cancer or disease.

Radford's opinion regarding an increased individual

ORDER RE SUMMARY JUDGMENT-   **155**

1 susceptibility factor of five will be stricken on <u>Daubert</u>

2 grounds. He will not be allowed to testify about individual

3 susceptibility. His opinion amounts to no more than subjective

4 belief and/or unsupported speculation. "Individual

5 susceptibility" does not appear to be a matter which Radford has

6 researched independent of this litigation. He has never analyzed

7 the range of susceptibility within a population. All indications

8 are that this was his first foray into this area. Radford's

9 analysis has not been subjected to normal scientific scrutiny

10 through peer review and publication. Although there is

11 acceptance within the scientific community that susceptibility

12 exists, there is no general acceptance that risk estimates should

13 be increased by a factor of five to account for such

14 susceptibility.

15   The real clincher, however, is Radford's failure to offer a

16 satisfactory explanation for ignoring Isselbacher regarding the

17 existence of individuals with lower susceptibility and his

18 failure to give that any consideration in his analysis of

19 susceptibility. It indeed suggests Radford's only consideration

20 was to **increase** risk estimates. The court is persuaded by the

21 argument that an increased individual susceptibility factor is

22 unnecessary due to the fact the risk estimates already take into

23 account a full range of susceptibilities.

24   Not only is Radford's individual susceptibility factor

25 "unreliable" under Prong 1 of <u>Daubert</u>. Testimony about

26 individual susceptibility is not helpful to a jury because of the

27 present reality that there is no way to identify persons who are

28

ORDER RE SUMMARY JUDGMENT-   156

1 allegedly more susceptible to radiation-induced thyroid cancer,

2 nor can alleged differences in susceptibility be quantified.

3 Such testimony is irrelevant and does not "fit" under Prong 2 of

4 Daubert.

5 Defendants' motion in limine will be granted insofar as

6 concerns Radford's analysis and conclusions about individual

7 susceptibility.[108] The result is that Radford's thyroid cancer

8 estimates and doubling doses will stay at 5 rads for those 0 to 4

9 at the time of exposure; 10 rads for those 5 to 9 at the time of

10 exposure; 33 rads for those 10 to 19 at the time of exposure; and

11 100 rads for those 20 and over at the time of exposure. These

12 are the doubling doses which will be used for summary judgment

13 purposes to infer that radiation exposure is a "more likely than

14 not" cause of an individual's thyroid cancer. Anyone who can

15 prove exposure in excess of these doses is entitled to have

16 his/her claim considered by a jury.[109]

17 ─────────────────────

18 [108] A section of Dr. Mayer's November 1995 report is
dedicated to "The Issue of Susceptibility and its Incorporation
into Radiation Risk Models." (Mayer 1995 Rpt. at pp. 12-13).

19 Mayer adopts "a mathematical approach to deal with this issue
using the concept of 'susceptibility' as operationalized and

20 estimated by Finkel." He concludes the calculations made by him
"are supportive of the susceptibility range presented by Dr.

21 Radford presented in his report." However, there is nothing in
Mayer's work which salvages Radford's increased susceptibility

22 factor. Furthermore, although Mayer may be qualified to
construct mathematical models, he is not qualified, like Radford,

23 to opine about "susceptibility" as a radiobiological matter.

24

25 [109] These doubling dose levels are lower than what
defendants say are the doubling doses derived from Dr. Clifton's

26 analysis- 50 rads for individuals 10-19 at time of exposure; 16
rads for individuals 0-9 at time of exposure (incorporating a

27 DREF of 0.66).

28 **ORDER RE SUMMARY JUDGMENT-   157**

1   Defendants can try to persuade a jury that Radford's equal

2   effectiveness opinion and his age adjustments are not correct and

3   therefore, that the doubling dose for a particular individual

4   with thyroid cancer should be lower. However, for purposes of

5   generic causation, Radford's risk estimates, incorporating his

6   age adjustments and his assumption of equal effectiveness between

7   internal and external radiation, will be used to determine which

8   thyroid cancer claims survive summary judgment.[110]

9

10   **c. Non-Neoplastic Thyroid Conditions**

11   Radford addresses several non-neoplastic thyroid conditions,

12   including hypothyroidism, autoimmune thyroid disease, Graves'

13   disease, hyperthyroidism and chronic thyroiditis. According to

14   Radford, radiation exposure is "associated" with these

15   conditions. (Radford 1995 Iodine Rpt. at p. 19).

16

17   _____

18

19   [110] Radford uses his thyroid cancer risk estimates for
thyroid nodules and adenomas. The defendants argue that these

20   conditions are not compensable as physical injuries. However,
that is not a causation question. If they are physical injuries,

21   it seems the same risk estimates should apply. Furthermore, in
the absence of a cognizable physical injury, there may still be

22   viable claims for emotional distress ("cancerphobia") based on
the mere presence of nodules and adenomas. However, a **reasonable**

23   fear of contracting thyroid cancer depends on the extent of the
actual risk a person has in contracting cancer. Therefore, a

24   person with nodules or adenomas who asserts a claim for
"cancerphobia" will still need to prove he was exposed to a dose

25   of Hanford I-131 which doubles his risk of getting thyroid
cancer.

26   Whether subclinical conditions constitute **physical** injuries
is discussed <u>infra</u>.

27
28   **ORDER RE SUMMARY JUDGMENT-   158**

(1)   Autoimmune thyroid disease and Graves' disease

In his report, Radford states that recent studies of groups irradiated for medical purposes show a "radiation dose-related excess" of autoimmune thyroid disease and Graves' disease.[111] The three studies cited by Radford are Kaplan, et al., 1988; Loeffler, et al., 1988[112]; and Hancock, et al., 1991.[113] According to Radford:

> Autoimmune thyroid disease occurs when antibodies to thyroid proteins circulate in the blood and cause thyroid inflammation. These effects can be serious and eventually may lead to hypo-thyroidism. Radiation is thought to damage the thyroid tissue and cause release of thyroid antigens which can stimulate the formation of anti-thyroid antibodies. Such antibodies are also associated with Graves' disease. Kaplan, <u>et al.</u>, 1988 concluded that radiation exposure was a risk factor for autoimmune thyroid disease at relatively low exposures to the thyroid, even when delivered at small doses over a period of time.

(Radford 1995 Iodine Rpt. at pp. 19-20).

In the "Causation" section of his report, Radford says:

> In the case of immune (sic) thyroid disease or hyperthyroidism, the evidence **is still not sufficiently quantitative to permit risk and causative dose estimates.** However, since the **association** has been established, any cases involving these diseases would therefore have to be analyzed on an individual basis in relation to [I-131]

---

[111]   His report does not offer a clinical description of Graves' disease, nor does he specify the conditions which he believes fall within the "autoimmune thyroid disease" category.

[112]   Loeffler, et al., "The Development of Graves' Disease Following Radiation Therapy in Hodgkin's Disease," 14 **International Journal Radiation Oncology Physics** 175 (1988). Defendants' Ex. 134.

[113] Hancock, et al., "Thyroid Diseases After Treatment of Hodgkin's Disease," 325 **The New England Journal of Medicine** 599 (Aug. 1991). Defendants' Ex. 135.

ORDER RE SUMMARY JUDGMENT-   159

1   exposure.

2 (Id. at p. 28) (Emphasis added).

3

4   **(a) Fit/Relevancy**

5   Plaintiffs' burden is to produce evidence of the doubling

6 dose at which it is reasonable to infer I-131 exposure is a "more

7 likely than not" cause of autoimmune thyroid disease or Graves'

8 disease. Without causative risk estimates, there can be no

9 doubling doses. Evidence that I-131 is merely "capable of

10 causing" a disease is insufficient to meet the "doubling of risk"

11 standard.[114]

12   Radford's opinion that there is a "causative association"

13 between I-131 and autoimmune thyroid disease and Graves' disease

14 does not "fit" the pertinent causation inquiry and therefore, is

15 inadmissible under Daubert's relevancy prong.

16

17   **(b) Reliability**

18   Defendants contend Radford's opinion that there is an

19 "association" between I-131 and autoimmune thyroid disease and

20 Graves' disease is not supported by scientifically valid

21 evidence.

22   Defendants note that an "association" does not necessarily

23 _____

24   [114] Had there been no specific generic causation stage established in this litigation, and had the court proceeded directly to individual cases, it would be appropriate for a jury

25 to hear evidence that I-131 is "capable of causing" the individual's disease. It would be relevant to the overall

26 causation analysis, even if by itself it could not sustain a jury verdict requiring a finding that I-131 is a "more likely than

27 not" cause of the individual's disease.

28 **ORDER RE SUMMARY JUDGMENT-   160**

1   imply a causal relationship. They contend the studies cited by

2   Radford in support of his conclusion that there is an

3   "association" between radiation and autoimmune thyroid disease

4   and Graves' disease do not establish the "association" is

5   "causal," because they do not satisfy the epidemiological

6   criteria necessary to infer causation: strength of association,

7   temporal relationship, consistency of the association, biologic

8   plausibility, consideration of alternative explanations,

9   specificity of the association, and dose-response relationship.

10 ("Reference Guide on Epidemiology" at pp. 160-61).

11   The first study is Loeffler 1988 which involved Hodgkin's

12 disease patients who received radiation doses to the thyroid

13 between 3600 and 4000 rads. Defendants argue that because

14 Loeffler is a high dose study, it is misleading for inferring

15 risk at lower doses. Defendants cite a portion of Radford's

16 deposition testimony in which he indicates that studies involving

17 therapeutic doses of radiation are misleading for inferring risk

18 at lower doses because such doses are "much higher." (Radford

19 Dep. at p. 376).

20   Secondly, defendants say Loeffler provides no evidence of a

21 "dose-related excess" because it did not even consider dose-

22 response relationships. As such, defendants assert Loeffler

23 "fails to meet two critical causation criteria and cannot support

24 Radford's opinion about an alleged association between radiation

25 and autoimmune thyroid disease or Graves' disease."

26   The second study is Hancock which also involved Hodgkin's

27 disease patients who received therapeutic radiation treatments

28 **ORDER RE SUMMARY JUDGMENT-   161**

1 resulting in thyroid doses between 750 and 4400 rads. As with

2 the Loeffler study, defendants claim the problem with the Hancock

3 study is it involved high doses which are misleading for

4 inferring risk at lower doses.

5   Plaintiffs contend Loeffler and Hancock support the

6 existence of a "general causal relationship" between thyroid

7 irradiation and Graves' disease.[115] They say Loeffler found a

8 "statistically significant" increase in the prevalence of Graves'

9 disease in the exposed population because the incidence of the

10 disease was five to six times that of the unexposed population.

11 According to plaintiffs, Hancock found a statistically

12 significant association between irradiation and the subsequent

13 development of hyperthyroidism and hypothyroidism, including

14 Graves' disease.

15   Defendants do not dispute Loeffler and Hancock reported such

16 findings. However, defendants' concern is with the use of high

17 dose studies to infer risk at **lower doses**. Defendants argue that

18 because Radford makes no effort to extrapolate the results of

19 these high-dose studies to the "minimal doses at issue here, much

20 less a scientifically valid effort, the studies are unreliable as

21 evidence and irrelevant to this case."

22   According to plaintiffs, Radford cites Loeffler and Hancock

23 for the **"general proposition"** of the **"causal association"** between

24 radiation and Graves' disease (i.e. that radiation is "capable of

25 _____

26   [115] At his deposition, Radford confirmed that the focus of
   the Loeffler and Hancock studies was Graves' disease, as opposed
27 to "autoimmune thyroid disease."   (Radford Dep. at p. 422).
28
   ORDER RE SUMMARY JUDGMENT-   162

1 causing" Graves' disease). Plaintiffs add the fact "[t]hat

2 neither study considered dose-response relationships, or other

3 potential indicators of causation, is utterly irrelevant here:

4 the studies both show a powerful relationship between irradiation

5 and the diseases at issue."

6   Defendants have not provided a compelling reason for finding

7 Radford's reliance on Loeffler and Hancock as "unscientific" for

8 the "general proposition" that there is a "causal association"

9 between radiation and Graves' disease, albeit at **high doses**.

10 Indeed, at his deposition, Radford admitted Hancock is not a

11 convincing study for inferring the risk of Graves' disease among

12 populations exposed to radiation at **low doses**. (Radford Dep. at

13 p. 420). According to Radford: "I thought I stressed in my

14 report that the evidence as far as Graves' disease is still

15 sufficiently **uncertain** that I did not feel it appropriate to

16 include risk coefficients for Graves' disease." (Id.)[116]

17   The controversy really centers around Radford's reliance on

18 the Kaplan study and his (Radford's) opinion that it shows

19 radiation exposure is a risk factor for autoimmune thyroid

20 disease at relatively **low exposures** to the thyroid. It appears

21 plaintiffs assert the same is true for Graves' disease because

22 that condition falls within the category of "autoimmune thyroid

23 disease" as used by Kaplan. Kaplan noted "[t]here was a trend

24 _____

[116] An opinion that I-131 is "capable of causing" Graves'
25 disease at high doses cannot prove that I-131 is a "more likely
than not" cause of Graves' disease. For that matter, neither can
26 an opinion that I-131 is "capable of causing" Graves' disease at
low doses.

27
28 **ORDER RE SUMMARY JUDGMENT-   163**

1   toward a higher frequency of autoimmune thyroid disease, either

2   Hashimoto's thyroiditis or previously treated **Graves' disease** in

3   the exposed group."   (Kaplan at p. 380).

4   Defendants contend Kaplan suffers from serious flaws in

5   "each and every one of the epidemiological evaluative criteria."

6   First of all, defendants note that the results Kaplan reported

7   for the autoimmune thyroid disease category at the 95% confidence

8   interval were not statistically significant. Although the

9   prevalence ratio (akin to relative risk) was 2.2, the range was

10   0.8 to 6.2 and therefore, included 1.0, the background incidence

11   of the disease.

12   At his deposition, upon examination by plaintiffs' counsel,

13   Radford asserted that 90% confidence intervals are "widely used,"

14   as opposed to 95% confidence intervals. Radford estimated that

15   if a 90% confidence interval were used, the range would be around

16   "1 to 5.8, perhaps." He asserted this would be "statistically

17   significant." (Radford Dep. at pp. 437-38). However, as

18   defendants note, even this confidence interval includes 1.0- the

19   background incidence- and therefore, is not statistically

20   significant. ("Reference Guide on Epidemiology" at pp. 154-55;

21   173).[117]

22   Plaintiffs argue that defendants' main critique of Kaplan is

---

[117]   "The width of the confidence interval provides an
indication of the precision of the point estimate or relative
risk found in the study; the narrower the confidence interval,
the greater the confidence in the relative risk estimate found in
the study. **Where the confidence interval contains a relative
risk of 1.0, the results of the study are <u>not statistically
significant</u>.**"   ("Reference Guide on Epidemiology" at p. 173)
(Emphasis added).

ORDER RE SUMMARY JUDGMENT-   164

1 that it is statistically significant only at the 90% confidence

2 range. Defendants say no such thing. They dispute statistical

3 significance even at the 90% confidence interval. Radford's

4 assertion of statistical significance appears to be an error on

5 his part.

6   The defendants level other criticisms at Kaplan regarding

7 "specificity," "dose level," and "dose-response relationship."

8 Defendant say Kaplan does not provide specific data for the

9 thyroiditis conditions that Radford contends fall within the

10 ambit of "autoimmune thyroid disease." They say Kaplan provides

11 no data about dose-response relationships and no basis for

12 analyzing such because it is merely a prevalence study. They

13 contend Kaplan did not account for thyroid doses in the "hundreds

14 of rads" received by some study subjects.

15   These criticisms were discussed _supra_ in conjunction with

16 the defendants' motion in limine against Dr. Ruttenber. For

17 essentially the same reasons offered there, the court does not

18 believe "specificity" or absence of "dose-response relationship"

19 are sufficient to discredit Radford's opinion that there is a

20 causal "association" between radiation and autoimmune thyroid

21 disease and Graves' disease. Those matters go to the "weight" of

22 the opinion and do not preclude admissibility.

23   The court concluded the existence of unaccounted doses in

24 the Kaplan study does not prevent Dr. Ruttenber from relying on

25 that study for the general proposition that I-131 is "capable of

26 causing" autoimmune thyroiditis. Ruttenber offers no opinion

27 about a particular dose. He just says that at some level, I-131

28 **ORDER RE SUMMARY JUDGMENT-   165**

1 is "capable of causing" autoimmune thyroiditis.

2   The same holds true here if all Dr. Radford is contending is

3 that in general I-131 is "capable of causing" autoimmune thyroid

4 disease and Graves' disease. However, Radford asserts Kaplan

5 stands for the proposition that radiation exposure is a risk

6 factor for autoimmune thyroid disease at relatively **low exposures**

7 to the thyroid. The existence of unaccounted for doses is

8 material to that proposition because it calls into question the

9 dose range reported by Kaplan (11 to 112 rads) and in turn

10 whether radiation exposure is indeed a risk factor for autoimmune

11 thyroid disease at that low of a range.

12   The plaintiffs contend Kaplan specifically addressed this

13 concern:

14   Radiation exposure from thyroid scintiscans
            before the study adds additional uncertainty
15   to the dose estimates but probably did not
            alter the study results markedly. It seems
16   likely that many of the scintiscans reflected
            thyroid disease present at the time of the
17   procedure [fluoroscopic examination]; for many
            of the women in both groups [control and exposed]
18   who had been scanned, the prior diagnoses reported
            by the woman [sic] were the same as the diagnoses
19   in Table 5.

20 (Kaplan at p. 380). Plaintiffs say the fact certain persons

21 within the control group also received unaccounted for doses

22 "counterbalances to a certain degree the potential effect on the

23 study group."

24   Plaintiffs omit the balance of the quote from Kaplan which

25 is what defendants focus on:

26   Radiation doses from iodine-123 and pertechnetate
            scans are low. **Scans performed with iodine-131**
27   **may have delivered dozens up to 200 rads to the**

28 **ORDER RE SUMMARY JUDGMENT-   166**

**thyroid, <u>but patients given diagnostic iodine-131</u>
<u>do not have increased rates of thyroid cancer.</u>**

(Kaplan at p. 380) (Emphasis added). At his deposition, Radford
said he disputed this rationale (that patients given diagnostic
iodine-131 do not have increased rates of thyroid cancer) and
therefore, if he had conducted the study, he would have excluded
the doses. Radford added that he would also have wanted a
control group which had not been exposed to any radiation.
(Radford Dep. at pp. 427-28).

Dr. Boice, the lead epidemiologist on the Kaplan study,
states that one of the "serious deficiencies in our study was our
inability to estimate reliable thyroid dose estimates associated
with the fluoroscopies." (Boice Affidavit at Paragraphs 22-23).
Boice concludes that due to this "deficiency" and others,
including lack of statistical significance and small size of the
study population, that "[i]t would not be appropriate to rely on
[the Kaplan study] to infer that radiation doses of up to 112
rads cause autoimmune thyroid disease or any of the conditions
included in this broad category." (<u>Id</u>. at Paragraph 37). He adds
that "we [he and Kaplan] were not willing to and did not conclude
that the study established a causal relationship between
autoimmune thyroid disease (or antibody-positive hypothyroidism,
antibody-negative hypothyroidism, thyroiditis, or Graves'
disease) and **low dose radiation**." (<u>Id</u>. at Paragraph 38)(Emphasis
added).

Without Kaplan, Radford has no basis whatsoever for
asserting a "generic causal association" between I-131 and

ORDER RE SUMMARY JUDGMENT-   167

1   autoimmune thyroid disease and Graves' disease at **low doses.**

2   Radford cannot rely on Kaplan for such a proposition, due in

3   particular to the lack of statistical significance and

4   uncertainty in the dose estimates.

5   This conclusion is not inconsistent with the court's

6   discussion regarding Ruttenber's use of Kaplan. Ruttenber does

7   not try to glean as much from Kaplan as Radford does.

8   Ruttenber's **report** does not assert that Kaplan establishes a

9   connection between low dose radiation and autoimmune thyroiditis.

10   He says it is only one piece of the puzzle (along with the high

11   dose Spitalnik study and the Nagataki study) establishing that at

12   some dose level, radiation is "capable of causing" autoimmune

13   thyroiditis. Radford, on the other hand, cites Kaplan as

14   supporting a generic causal association between autoimmune

15   thyroid disease and low doses of radiation and goes so far as to

16   assert that one of the reasons is the existence of statistical

17   significance at the 90% confidence interval.

18   While there may be some genuine scientific debate whether

19   Kaplan, along with other studies, supports the notion of a causal

20   association between I-131 and autoimmune thyroid disease at **some**

21   **level,** it does not support the notion of such an association at

22   **low level exposure.**

23   Radford essentially admitted as much at his deposition. He

24   was asked to comment on the following statement of the Kaplan

25   study authors that:

26   . . .a definitive study was not practical
             in this population because of the large number
27   of subjects needed, but it would be of considerable
28
     ORDER RE SUMMARY JUDGMENT-   168

1   interest to learn whether **there is an association
           of low level radiation exposure to the thyroid with
2   clinically significant autoimmune thyroid disease.**

3   (Kaplan at p. 381) (Emphasis added). Radford's response was that

4   "they did show **some** relationship to autoimmune thyroid disease .

5   . . . they found **some** evidence of autoimmune disease related to

6   radiation exposure." (Radford Dep. at pp. 431-32)(Emphasis

7   added). He added:

8   [W]hether this study [Kaplan] **by itself** proves
           the point is dependent on the various limitations
9   that it had. . . . [O]bviously, a **body of
           scientific information** is developing which does
10   **suggest a relationship** between autoimmune thyroid
           disease and radiation exposure.
11
    (Id. at 432)(Emphasis added). Radford acknowledged that the
12
    "relationship" is not established for certain and "warrants
13
    further study." (Id.)
14
          Radford's opinion about **low dose** causal association between
15
    I-131 and autoimmune thyroid disease (including Graves' disease)
16
    is not scientifically reliable. However, it is not
17
    scientifically unreliable for Radford to opine, based on the
18
    Kaplan, Loeffler and Hancock studies as a whole, that at least at
19
    high dose levels, exposure to I-131 is "capable of causing"
20
    autoimmune thyroid disease and in particular, Graves'
21
    disease.[118]
22
    _____

23   [118] Of course, this does not mean the court is finding **as a
    matter of law** that radiation exposure is "capable of causing"
24 autoimmune thyroid disease at some level. In attacking the
    "weight" of such an opinion, the defendants could cite Dr.
25 Ruttenber's equivocal opinion that based on Loeffler and Hancock
    (high dose external radiation studies), there is sufficient
26 epidemiologic evidence to only "suspect" a causal relation at
    doses from external radiation above 750 rads. (Ruttenber Dep. at
27 p. 173). According to Ruttenber:
28
    ORDER RE SUMMARY JUDGMENT-   169

1 **(2) Hyperthyroidism**

2 **(a) Fit/Relevancy**

3 As noted above, Radford acknowledges the data is

4 insufficient to permit calculation of risk and causative dose

5 estimates for hyperthyroidism. Accordingly, for the same reasons

6 specified above in the discussion of autoimmune thyroid disease

7 and Graves' disease, Radford's opinion that I-131 is "associated"

8 with hyperthyroidism does not fit the relevant causation inquiry-

9 at what dose level can I-131 be considered a "more likely than

10 not" cause of hyperthyroidism (i.e. at what level does the risk

11 double?).

12 Plaintiffs cannot sustain their ultimate burden of proof on

13 the basis of Radford's opinion. As such, Radford's opinion is

14 not helpful to the jury and will be stricken on the basis of

15 _Daubert's_ fit/relevancy prong. An opinion that radiation is

16 "associated" with hyperthyroidism only allows speculation on the

17 part of a jury as to whether radiation is a "more likely than

18 not" cause of hyperthyroidism in a particular individual.

19

20 **(b) Reliability**

21 Defendants, however, also challenge the scientific

22 _____

23 [W]e might suspect a relation. It's been found
   in these reports [Loeffler and Hancock], the idea
24 about the mechanism of Graves' disease is one that's
   similar to autoimmune disease in general, but there
25 are very few reports.

26 (_Id_.)
   Because Loeffler and Hancock are external radiation dose
27 studies, there would obviously be a DREF issue here as well.

28 **ORDER RE SUMMARY JUDGMENT-   170**

reliability of Radford's opinion that radiation is even

"associated" with hyperthyroidism.   In his report, Radford

stated:

> The thyroid abnormalities associated with
> radiation exposure in addition to thyroid
> cancers include: solitary thyroid nodules,
> thyroid adenomas, hypothyroidism, autoimmune
> thyroid disease, **and possibly hyperthyroidism.**
> (Yoshimoto, et al., 1995; Conard, et al., 1966;
> Tamura, et al., 1981; Katayama, et al., 1985).

(Radford 1995 Iodine Rpt. at p. 15) (Emphasis added).

At his deposition, Radford said he used the word "possibly"

because the association was not "so clearly indicated." Radford

testified that he based his opinion on one report- Katayama.[119]

Specifically, Radford referred to Table 2 of Katayama which he

said lists seven patients who developed hyperthyroidism at

thyroid doses ranging from 30 to 3,000 rads, but "[m]ostly in the

30, 40, 100, 200 range." Radford testified he was not aware of

any report, other than Katayama, reporting an association between

radiation and hyperthyroidism. (Radford Dep. at pp. 318-20).

Defendants contend Radford cannot premise on Katayama an

opinion about a causal association between radiation and

hyperthyroidism. According to defendants, Katayama involved

patients who were treated with **high doses** of external radiation

for gynecological malignancies. Of 1269 patients whose records

were examined, five who had received a tumor dose ranging from

5,000 to 8,000 rads to the abdomen and/or pelvis developed

---

[119] Katayama, et al., "Radiation Associated Hyperthyroidism in Patients with Gynecological Malignancies," 16 **Journal of Medicine** 588 (1985). Defendants' Ex. 60.

ORDER RE SUMMARY JUDGMENT-   171

1   hyperthyroidism between 2 and 8 years following their treatment.

2   (Katayama at p. 592).

3   Furthermore, defendants contend that because the authors of

4   the Katayama study do not define "hyperthyroidism," it is unclear

5   whether the study addresses the "specific" disease for which

6   Radford offers an opinion. Another deficiency of Katayama, say

7   defendants, is that it does not analyze dose-response

8   relationships between various doses of radiation and

9   hyperthyroidism. Defendants assert that Katayama fails the

10   "consistency" criterion because it is the only study dealing with

11   hyperthyroidism.   They also observe that Radford testified the

12   treatment the control group received is relevant in assessing the

13   value of the Katayama study, but the study "surprisingly" failed

14   to indicate how Katayama's patients were treated. (Radford Dep.

15   at p. 415). Therefore, say defendants, Radford cannot tell

16   whether the manner of treatment was a potential confounding

17   factor or source of bias in the study. For all the foregoing

18   reasons (absence of specificity, absence of dose-response

19   relationship, absence of consistency, etc.), defendants contend

20   Katayama does not meet the criteria for assessing causal

21   relationships between radiation and thyroid disease.

22   Plaintiffs respond to defendants' "specificity" challenge by

23   noting that Katayama refers to "Graves' disease associated with

24   prior thyroidal irradiation manifested by either hyperthyroidism

25   and/or ophthalmopathy." (Katayama at p. 588). However, in his

26   report, Radford treated Graves' disease and hyperthyroidism as

27   separate conditions. (Radford 1995 Iodine Rpt. at 19). The

28   **ORDER RE SUMMARY JUDGMENT-   172**

court cannot locate a specific definition of hyperthyroidism in Radford's report. At his deposition, Radford stated that Katayama related hyperthyroidism to an "auto-immune response." (Radford Dep. at pp. 319-20). In his declaration, Radford states the clinical conditions associated with autoimmune diseases of the thyroid are "a) Graves' disease **usually involving hyperthyroidism,** b) thyroiditis and c) Hashimoto's disease." (Radford Declaration at Paragraph 1; Ex. 7 to Plaintiffs' Appendix 1 re Iodine Claims) (Emphasis added). However, he still does not define hyperthyroidism.   It appears that between Radford's declaration and plaintiffs' response brief, plaintiffs are contending hyperthyroidism falls within the autoimmune thyroid disease category. Therefore, they argue a causal association between hyperthyroidism and radiation is supported by the same studies which, according to them, show a causal association between radiation and Graves' disease, and radiation and autoimmune thyroid disease in general (i.e. Hancock and Kaplan).[120]

Radford never made such a specific assertion in either his report or at his deposition. In his report and at his deposition, he said he relied **only** on Katayama for an association between radiation and hyperthyroidism. Furthermore, in his report, Radford stated that in the case of **"immune thyroid disease or hyperthyroidism,** the evidence is still not

---

[120] Where conditions do not exactly have the same etiology, but are the same **type** of disease (autoimmune type of disease), the "specificity" requirement is met. "Reference Guide on Epidemiology," at p. 153.

ORDER RE SUMMARY JUDGMENT-   173

1 sufficiently quantitative to permit risk and causative dose

2 estimates." (Radford 1995 Iodine Rpt. at p. 28)(Emphasis added).

3 On its face, this indicates Radford is treating autoimmune

4 thyroid disease and hyperthyroidism as separate entities. At p.

5 15 of his report, Radford also separates out "autoimmune thyroid

6 disease" from "hyperthyroidism." From all of this, the court

7 finds it is indeed unclear whether Katayama is using the same

8 definition of hyperthyroidism as Radford.

9   Plaintiffs contend the defendants dishonestly attempt to

10 portray Katayama as a "high dose" study. Plaintiffs note that

11 while the doses to the abdomen and/or pelvis were "comparatively

12 high," the relevant radiation dose to the thyroid was estimated

13 to be in the range of 30 to 200 rads. According to plaintiffs,

14 this establishes a "causal association between **low-dose** thyroid

15 irradiation and hyperthyroidism." (Emphasis added).

16   Nowhere in his report, at his deposition, or even in his

17 declaration, does Radford specifically assert that Katayama (or

18 anything else) supports a causal association between **low-dose**

19 thyroid irradiation and hyperthyroidism. Katayama certainly did

20 not reach such a conclusion. According to Katayama:

21   In 5 out of 1,269 patients with radiation
           therapy, hyperthyroidism developed 14 months
22   to 88 months after irradiation with the tumor
           dose ranging from 5,000 to 8,000 rads to
23   the abdomen and/or pelvis. . . . **These cases
           were calculated to have had 30 to 200 rads to
24   the thyroid except one case in which the exact
           data were not available.**

25 (Katayama at p. 592)(Emphasis added).

26   The thyroid doses were part of the **overall** dose.

27

28 **ORDER RE SUMMARY JUDGMENT-   174**

1 Plaintiffs' lawyers (not Radford) extract the thyroid dose (30 to

2 200 rads) and assert that Katayama found a causal association

3 between that dose and hyperthyroidism. That is false. Rather,

4 Katayama found:

> . . . our present study confirmed that **abdominal
> and/or pelvic irradiation <u>may</u> be associated
> with later onset of hyperthyroidism, even if <u>its</u>
> thyroidal dose is very low.** The patients who had
> irradiation **to the abdomen and/or pelvis** should
> be carefully followed because of a higher risk
> of developing thyroidal pathology and dysfunction,
> including not only neoplasms and hypothyroidism but
> also hyperthyroidism.

10 (Katayama at pp. 593-94)(Emphasis added). Elsewhere, Katayama

11 emphasizes the present study was "designed to clarify the

12 significance of **abdominal irradiation** [not thyroidal irradiation]

13 in the later onset of hyperthyroidism." (<u>Id</u>. at pp. 592-

14 93)(Emphasis added). Katayama did not analyze the thyroid doses

15 separately nor assert there was a causal association between

16 those doses and hyperthyroidism.

17   In sum, the court agrees with defendants that Katayama is

18 essentially a high dose study. On the other hand, the fact

19 Katayama does not analyze dose-response relationships should not,

20 by itself, bar its use for inferring a causal association between

21 radiation and hyperthyroidism, but most assuredly not at low

22 doses. The existence of a dose-response relationship is not

23 necessary to infer causation. ("Reference Guide on

24 Epidemiology," at p. 164). The defendants do not offer any

25 reason why there should be a dose-response relationship between

26 radiation and hyperthyroidism.

27   Insofar as "consistency," it is indeed true that Katayama is

28 **ORDER RE SUMMARY JUDGMENT-   175**

the only study on which Radford hangs his hat. Radford's
assertion of a causal association would be bolstered if he could
cite other studies replicating the Katayama results. On the
other hand, defendants have not pointed to any other studies
which are **inconsistent** with the Katayama results. "Different
studies that examine the same exposure-disease relationship
should yield similar results. Any inconsistencies signal a need
to question whether the relationship is causal." ("Reference
Guide on Epidemiology," at p. 162).

The court does not find compelling the defendants' argument
that because Katayama did not say how its control group was
treated, Radford cannot say whether the manner of treatment was a
potential confounding factor. Plaintiffs acknowledge Katayama
compared its exposed group to an "imperfectly characterized
control group," but they assert it corrected for this "potential
weakness" by comparing the study group findings to the results of
a general population study (Furszyfer, et al. (1972)). The
defendants offer no reply to that argument. In any event,
because it is not established that there was actually a
confounding factor that should have been considered, the
defendants' argument, at most, goes to the "weight" that should
be accorded to the Katayama study and in turn, the "weight" to be
accorded Radford's opinion. The existence of a **potential**
confounding factor, by itself, is not sufficient to preclude an
inference that radiation is associated with hyperthyroidism.

In his report, Radford says nothing specific about the
"biological plausibility" of radiation causing hyperthyroidism.

ORDER RE SUMMARY JUDGMENT-   176

1 However, at his deposition, Radford asserted:

2   Well, on the basis of the pathogenesis of
             a variety of thyroid diseases, I think it is

3   a reasonable biological inference that radiation
             can cause hyperthyroidism, and I have relied on

4   Doctor Peters' report in this case to indicate
             the latest thought in the area.

5

6 (Radford Dep. at p. 321).

7   Dr. Sara Peters, a pathologist, has prepared a report on

8 behalf of the plaintiffs which is the subject of a motion in

9 limine addressed <u>infra</u>. Radford acknowledged he did not cite Dr.

10 Peters in his report and that he only spoke to her three or four

11 days before his deposition. He spoke with her by telephone from

12 the office of plaintiffs' counsel for a total of fifteen to

13 twenty minutes. Radford said he did not cite Dr. Peters in his

14 report because at that time, he "didn't know Dr. Peters was in

15 the case." (Radford Dep. at pp. 321-22). According to Radford:

16   . . . I felt it was important to talk to
             a practicing thyroidologist, a person who

17   was working in the field of thyroid abnormalities,
             as to what the latest views were on possible

18   effects of radiation, as well as other pathologic
             conditions in the thyroid.

19 (Radford Dep. at p. 322).

20   The defendants say Dr. Peters' report contains no analysis

21 of hyperthyroidism. They argue that if it was so "important" for

22 Radford to talk to a practicing thyroidologist, he should have

23 done so before he submitted his reports. Furthermore, they

24 contend Radford has no basis for relying on Dr. Peters as a

25 "reliable source of objective information about thyroid disease"

26

27
28 **ORDER RE SUMMARY JUDGMENT-**   **177**

1 solely on the basis of a short telephone call.[121]   Defendants

2 contend Radford's reliance on Dr. Peters is yet another example

3 of him offering an opinion first, and then seeking support for it

4 later.   Such "methodological fluctuations", argue defendants, are

5 unscientific.

6   Plaintiffs say Radford relied on Dr. Peters merely to

7 corroborate a conclusion at which he (Radford) had previously

8 arrived (biological plausibility).   Plaintiffs assert this was

9 reasonable for him to do and that "[w]here and when such

10 consultation takes place, and how long it lasts are not really

11 relevant criteria for judging sufficiency of evidence."

12 Plaintiffs assert that in her report, Dr. Peters includes

13 hyperthyroidism under the "rubric of autoimmune thyroid disease."

14 According to plaintiffs, Radford relies on his knowledge of

15 biological mechanisms, "his conversations with one of the

16 country's leading experts on the thyroid,"[122] and Dr. Peters for

17 his "biological mechanism conclusion."   Plaintiffs say that

18 because of Radford's background and credentials, he is qualified

19 to opine about this causal mechanism.

20   The court is not impressed with Radford's belated reliance

21 on Peters, especially so in light of his (Radford's) written

22 _____

23 [121]   At his deposition, Radford said he concluded Doctor
Peters was a "reliable source of independent information
concerning thyroid disease."   (Radford Dep. at p. 363).

24

25 [122]   Radford testified that on various occasions he speaks to
one of his medical school classmates- Milton Humolsky- a "leading
expert" on thyroid disease who tells him "what's latest in

26 thyroid."   (Radford Dep. at p. 359).   Radford does not elaborate
on what Humolsky has told him, in particular about radiation and

27 hyperthyroidism.

28 **ORDER RE SUMMARY JUDGMENT-   178**

report which says there is only a "possible" association between radiation and hyperthyroidism; refers separately to Graves' disease, autoimmune thyroid disease and hyperthyroidism; and says nothing about the biological mechanism by which radiation specifically causes hyperthyroidism. It appears proper to include Graves' disease and hyperthyroidism under the same autoimmune disease umbrella and indeed, they may share a similar autoimmune biological mechanism. However, the court cannot find where Radford describes hyperthyroidism as such in his reports. The court does not see where in his reports, Radford discusses hyperthyroidism under the "rubric of autoimmune thyroid disease." In her report, Dr. Peters refers to hyperthyroidism, but appears to consider it as synonymous with Graves' disease (Peters 1995 Rpt. at p. 6). In his reports, Radford refers **separately** to Graves' disease and hyperthyroidism.

Based on the foregoing, the court concludes Radford's use of Dr. Peters' report is a belated attempt to shore up a written report which stated there was only a "possible" association between radiation and hyperthyroidism. Perhaps recognizing the weakness for inferring causation in terms of specificity, dose-response relationship and consistency, Radford, at the last minute, turned to "biological plausibility" to bolster his opinion. The circumstances of Radford's reliance on Peters do not reflect the careful attention of a scientist to his methodology **before** he renders an opinion.

Like Ruttenber, Radford is bound to what he opines in his report. The requirement of exchanging experts report is to

ORDER RE SUMMARY JUDGMENT-   179

1 insure there will be no "moving targets." In his report, Radford

2 says only that there is a "possible" association between

3 radiation and hyperthyroidism. This is perhaps an implicit

4 recognition of the limitations of the Katayama study. As noted,

5 even an association does not necessarily imply a causal

6 relationship. As such, a "possible" association is nowhere close

7 to a causal relationship.

8   Radford has not offered a scientifically reliable opinion

9 that radiation is even "capable of causing" hyperthyroidism.

10 Consequently, there is no way plaintiffs can prove radiation is

11 a "more likely than not" cause of their hyperthyroidism.

12

13   **(3) Hypothyroidism and Chronic Thyroiditis**

14   Radford asserts radiation exposure is "associated" with

15 **"hypothyroidism**, autoimmune thyroid disease, Graves-disease,

16 hyperthyroidism and **chronic thyroiditis."** (Radford 1995 Iodine

17 Rpt. at p. 19). This is the only specific reference to "chronic

18 thyroiditis" in Radford's 1995 report. There is no specific

19 reference to that condition in Radford's 1996 supplemental iodine

20 report.

21   No scientific literature is cited by Radford in his reports

22 as supporting the "association" between radiation exposure and

23 chronic thyroiditis. The list of thyroid abnormalities found at

24 page 15 of Radford's 1995 iodine report does not include chronic

25 thyroiditis. The abnormalities listed are "solitary thyroid

26 nodules, thyroid adenomas, hypothyroidism, autoimmune thyroid

27 disease, and possibly hyperthyroidism." With regard to those

28 **ORDER RE SUMMARY JUDGMENT-   180**

1   conditions, Radford cites the following studies: Yoshimoto, et

2   al., 1995[123]; Conard, et al., 1966; Morimoto, et al., 1987[124];

3   Tamura, et al., 1981[125]; and Katayama, et al., 1985. (Radford

4   1995 Iodine Rpt. at p. 15).

5   At his deposition, Radford had difficulty identifying the

6   studies which supported the conclusion in his report that there

7   is an association between radiation and chronic thyroiditis. He

8   acknowledged Yoshimoto did not find a statistically significant

9   association between radiation exposure and chronic thyroiditis.

10 All Radford could say about Yoshimoto was that "they did find 2

11 cases [of chronic thyroiditis], **whatever that means."** (Radford

12 Dep. at pp. 411-12)(Emphasis added). Radford acknowledged the

13 Morimoto study did not observe any specific relationship between

14 development of chronic thyroiditis and radiation exposure.

15 (Radford Dep. at pp. 413-14).[126] Radford acknowledged the

16 Tamura study involved several thousand rad exposures (2,000 to

17 4,000 rads). (Radford Dep. at p. 414).

18 _____

[123] Yoshimoto, et al., "Prevalence Rate of Thyroid Diseases
19 Among Autopsy Cases of the Atomic Bomb Survivors in Hiroshima,
1951-1985," **Radiation Research** (1995). Defendants' Ex. 130.
20
[124] Morimoto, et al., "Serum TSH, Thyroglobulin, and
21 Thyroidal Disorders in Atomic Bomb Survivors Exposed in Youth:
30-Year Follow-up Study," 28 **Journal of Nuclear Medicine** 1115
22 (July 1987). Defendants' Ex. 83.

[125] Tamura, et al., "Thyroid Abnormalities Associated with
23 Treatment of Malignant Lymphoma," 47 **Cancer** 2704 (June 1981).
24 Defendants' Ex. 115.

[126] According to Radford, Morimoto found two cases of
25 chronic thyroiditis in the "exposed and 5 cases in the unexposed;
the exposed being **greater than 100 rads."** Radford asserted
26 "[t]hat sounds to me like there was an excess." (Radford Dep. at
27 p. 413)(Emphasis added).
28
ORDER RE SUMMARY JUDGMENT-   181

1   Plaintiffs contend defendants have artificially separated

2 the discussion of hypothyroidism and chronic autoimmune

3 thyroiditis. Plaintiffs assert that "[a]s hypothyroidism is a

4 subset of chronic autoimmune thyroiditis, and the most common

5 manifestation of it, studies that support a relationship with one

6 condition also support the other." In that regard, plaintiffs

7 refer to the Nagataki and Kaplan studies.

8   While there appears to be no dispute that chronic

9 thyroiditis can result in hypothyroidism (biochemical or

10 clinical)[127], the problem is that nowhere in either his original

11 report or his supplemental iodine report, or even at his

12 deposition, did **Radford** explain how Nagataki and Kaplan support

13 his opinion that there is an association between radiation

14 exposure and chronic thyroiditis. Without such an explanation,

15 it is insignificant that Nagataki and Kaplan are included in the

16 bibliography attached to Radford's original report.

17   In comparison, Dr. Ruttenber explained in his report and at

18 his deposition why he thought Kaplan and Nagataki supported the

19 existence of a causal association between radiation exposure and

20 chronic autoimmune thyroiditis. Ruttenber's conclusion that

21 there is a causal association is not methodologically bankrupt.

22 However, Ruttenber, **in his report**, limits himself to the general

23 proposition that I-131 is "capable of causing" chronic

24 thyroiditis, **not that it is "capable of causing" it at low doses.**

25 Radford does not say anything more than Ruttenber (i.e. that

26 _____

27   [127] See Ruttenber 1995 Iodine Rpt. at p. 16.

28 **ORDER RE SUMMARY JUDGMENT-   182**

1 there is a causal association between radiation and chronic

2 thyroiditis), however Ruttenber at least explains the methodology

3 behind his opinion. Most certainly, Radford cannot be used as

4 support for the proposition that there is a **low dose** causal

5 association between radiation and chronic thyroiditis.[128]

6   Radford's reports (original and supplemental) do say quite a

7 bit more about the association between radiation and

8 hypothyroidism. In his original iodine report, Radford states:

9   Hypothyroidism is a disease leading to a
           reduced thyroid function of varying severity,
10   which can seriously affect both the physical and
           psychological condition of the affected person.
11   One of the causes of hypothyroidism is radiation
           exposure (Maxon, 1985). Most of this literature
12   involves exposures as a result of medical
           treatment, including exposure to [I-131].
13   Additionally, hypothyroidism has been established
           as a result of exposure to radioiodine among
14   the Marshall Islanders exposed to atomic bomb
           tests (Conard, 1984; Simon, et al., 1993),
15   **at lower doses.**

16 (Radford 1995 Iodine Report at p. 19) (Emphasis added).

17   Radford adds:

18   Hypothyroidism has also been shown to be
           produced by radiation exposure, and indeed
19   high doses of radiation have been used to
           ablate thyroid function for medical reasons.
20   Results of investigations of the Marshall
           Islanders with regard to hypothyroidism
21   have been summarized above, and lead to
           the conclusion that even **low doses** can
22   result in depressed thyroid function. Although
           there are several studies showing hypothyroidism
23   at high doses (Hancock, et al., 1995), the
           exact dose-response relationship has not yet
24   been fully worked out. **It is possible that a**

25 _____

    [128] Like Radford, Ruttenber offers no quantitative risk
26 estimates with regard to chronic thyroiditis and does not
    identify the dose level at which I-131 exposure doubles the risk
27 of chronic thyroiditis.

28
    ORDER RE SUMMARY JUDGMENT-   183

**threshold for radiation required to produce
hypothyroidism is necessary, perhaps at about
20 rem (Maxon, <u>et al.</u>, 1977). At thyroid doses
above this level there is a reasonable likelihood
that hypothyroidism can be ascribed to [I-131]
exposure from Hanford, although the risk
coefficient cannot be precisely stated at this
time. Clinical factors, such as age at diagnosis,
could also be important in assigning causation.
Causation in these cases can only be analyzed on
an individual basis.**

(Radford 1995 Iodine Report at p. 27)(Emphasis added).

In his March 1996 supplemental iodine report, Radford includes a discussion of "Evidence for effects of **low doses** or radiation in producing hypothyroidism in the Marshall Islands." (Supp. Rpt. at pp. 4-7).

The bone of contention is not whether I-131 is "capable of causing" hypothyroidism. Defendants concede that much by their willingness to accept the doubling doses for hypothyroidism offered by Dr. Ruttenber, provided those doses are increased to account for a DREF for internally deposited I-131. Rather, defendants contend the scientific literature does not support Radford's assertion that I-131 is "capable of causing" hypothyroidism at **low doses.**

One of the studies Radford relies on for this assertion is Simon, et al., 1993.[129] Radford explains in his March 1996 supplemental report that this study involved an examination of 1300 Ebeye residents for thyroid abnormalities. Ebeye is one of the Marshall Islands. 1,050 of the 1,300 Ebeye residents were tested for TSH (thyroid stimulating hormone). According to

---

[129] Simon, et al., "Report on the Medical Findings of the Thyroid Disease Study in Ebeye," (1993). Defendants' Ex. 113.

ORDER RE SUMMARY JUDGMENT-   **184**

1 Radford, 30 were found to have elevated TSH levels, 8 had "very

2 high levels, and only two had previous thyroid surgery. (Supp.

3 Rpt. at p. 5). Radford went on to explain why he believed the

4 residents of Ebeye received thyroid doses approximately one-half

5 of those received by the residents of Utirik, an island much

6 closer to the atomic bomb test sites. He also offered reasoning

7 why he believed the Ebeye population would not have included

8 former residents of Utirik (who would have been exposed at the

9 time of their residency on Utirik). He asserted that exposure in

10 the "more remote atolls," such as Ebeye, "would have been

11 dominated by [I-131]." (Id. at pp. 5-7). Radford concluded:

> From considerations described above, we
> have strong evidence that hypothyroidism
> can be the result of low thyroid doses
> primarily from [I-131] evidently at thyroid
> doses less than half the doses to the Utirik
> study group. It is reasonable to extrapolate
> the higher dose results to the lower dose
> range to obtain a dose-response relationship
> for hypothyroidism and radiation exposure.
> The Marshall Island data do indicate that
> **mild hypothyroidism** can result from relatively
> low doses.

(Id. at p. 7)(Emphasis added).

The defendants assert there are numerous problems with the

Simon study. They claim the study did not find any excess of

hypothyroidism. They note Simon reported that "only 30" study

participants were found to have TSH levels "significantly

elevated to suggest subclinical hypothyroidism." (Simon 1993 at

p. 5). Defendants say a second problem with Simon is that it did

not report actual TSH values and therefore, Radford is in no

position to assess the significance of the elevated TSH readings

ORDER RE SUMMARY JUDGMENT-   185

1 as reflecting a hypothyroid condition. Finally, defendants

2 contend Radford did not know the residence history of 24 of the

3 30 residents with elevated TSH readings; the radiation doses that

4 these residents received; or how the TSH levels of the 30

5 residents compared with those of islanders who had not been

6 exposed to radioactive fallout.

7   At his deposition, Radford testified it was no longer his

8 assumption that the Simon study established an increased

9 incidence of hypothyroidism. According to Radford:

10   . . . on the basis of my assessment of the
                 Simon study, I'm not convinced that the
11   elevated TSH values that were reported in
                 the Simon study necessarily were significantly
12   different in the exposed group compared to
                 the nonexposed group.
13
                 . . . we were able to get additional
14   information on the individuals in the
                 study population, and as I say, the proportion
15   of elevated TSH values in the exposed group
                 was not greatly different. It was slightly
16   higher, but not greatly different from the
                 unexposed group, that is those who were born
17   after the testing was over.

18 (Radford Dep. at pp. 398-400).

19   Radford indicated he received this information after he

20 submitted his supplemental report, but

21 did not discuss with plaintiffs' counsel whether it would be

22 appropriate to submit another supplemental report advising of the

23 change. (Id. at pp. 401-02). Defendants assert this shows that

24 Radford abandoned the basis for his opinion when it was

25 contradicted by the facts, "then tried to bury the contradiction

26 by failing to inform defendants of the new information."

27   Plaintiffs acknowledge Radford reversed course on the Simon

28 **ORDER RE SUMMARY JUDGMENT-   186**

study, but they contend his subsequent explicit disavowal of the study indicates he is a "careful and conservative" scientist, rather than one who tried to keep the new information from the defendants. The court can only guess at Radford's motivations, but the critical point is that there is no doubt the Simon study does not support a causal association between low dose radiation and hypothyroidism. Therefore, the question is whether there is anything else to support that proposition.

The other study cited by Radford in his original and supplemental iodine reports is Conard, et al, 1984.[130] This study analyzed the TSH levels of 164 Utirik residents. TSH levels above 3 microunits per milliliter were considered "suggestive" of thyroid hypofunction. Above 6 microunits per milliliter, the results were considered "positive." According to Radford:

> In the 164 residents on Utirik, where thyroid doses range from 30 to 95 rads, there was only one positive case found, but the rate was still high for this small population. In addition, there were seven cases on Utirik with suggestive hypothyroidism. Among the much larger control group, there were only four observations of positive or suggestive hypofunction. Thus, at this stage of the investigation, it was clear that thyroid doses well below 100 rads were associated with evidence of hypofunction.

(Radford Supp. Rpt. at p. 5).

Defendants assert there are several problems with the Conard

---

[130] Conard, et al., **Late Radiation Effects in Marshall Islanders Exposed to Fallout 28 Years Ago, in Radiation Carcinogenesis: Epidemiology and Biological Significance**, (John D. Boice, Jr. and Joseph F. Fraumeni, Jr. eds.)(1984). Defendants' Ex. 18.

ORDER RE SUMMARY JUDGMENT-   187

study: 1) it did not indicate whether any of the "suggestive"

cases was ever confirmed as hypothyroidism, and at his

deposition, Radford indicated he had no reason to believe

otherwise (Radford Dep. at p. 391); 2) at his deposition, Radford

acknowledged a TSH reading above 3 microunits per milliliter does

not "in general" permit a diagnosis of hypothyroidism (Id.); and

3) even a reading above 6 microunits per milliliter does not

compel a diagnosis of **clinical** hypothyroidism. As such,

defendants claim Conard fails the "specificity" criterion.

The plaintiffs contend this criticism is based on the fact

Conard employs "sensitive" laboratory criteria (laboratory

measurements of TSH level) which represent subclinical or

biochemical hypofunction. (Conard at p. 61). The fact Conard

deals with "subclinical" or "biochemical" hypothyroidism does not

mean it flunks the "specificity" criterion. Certainly, that may

limit its usefulness insofar as evaluating the causal association

between radiation and **clinical** hypothyroidism. In his reports,

Radford is not explicit about whether his conclusions pertain to

"clinical" or "subclinical" hypothyroidism, although in his

supplemental report he refers to "mild" hypothyroidism. In their

response brief, plaintiffs suggest that what Radford is referring

to is "delayed onset" hypothyroidism which can occur at lower

doses as opposed to hypothyroidism which presents itself after

irradiation at higher doses.

At his deposition, Radford testified a TSH reading above 6

microunits per milliliter was "strong evidence" of thyroid

hypofunction, "[b]ut it is a gradation." (Radford Dep. at p.

ORDER RE SUMMARY JUDGMENT-   188

392)   He added that a reading of greater than 6 did not

"necessarily" amount to clinical hypothyroidism (Id. at p. 395);

that he did not know the extent of the relationship between

clinical signs and TSH levels (Id. at 392); and that there was no

statement in Conard that anybody on Utirik had clinical

hypothyroidism (Id. at 396).   Radford's deposition testimony

evidences his awareness of the limitations of Conard insofar as

drawing an inference about causal association between radiation

and clinical hypothyroidism.   However, it appears the same is not

true regarding subclinical or biochemical hypothyroidism.[131]

Plaintiffs acknowledge that Conard's criteria for

identifying "suggestive" and "positive" cases of hypothyroidism

are imperfect, but they claim this does not invalidate the

"power" of the study.   They say the matter goes to the "weight"

to be accorded the study, not its admissibility.   The court

agrees and notes defendants did not revisit the "specificity"

argument in their reply brief.

The argument defendants do revisit in their reply brief is

the one which points out the deficiency of Conard, et al., 1984,

as evidence of a causal association between **low dose** radiation

and subclinical or biochemical hypothyroidism.   The Utirik

thyroid doses reported in Conard, et al., were 30 to 95 rads.

However, a reanalysis of those dose levels was already being

undertaken at the time Conard, et al., 1984 was published.

Conard acknowledged this:

---

[131]   The compensability of subclinical hypothyroidism is
discussed infra.

ORDER RE SUMMARY JUDGMENT-   189

> Reevaluation of early dosimetry, now under way
> at Brookhaven National Laboratory with additional
> data that have become available, indicates that
> thyroid doses may be **higher** than previously estimated.

(Conard, et al. 1984 at p. 58, n. 1). Radford testified he did

not make any effort to determine whether the doses had been

reanalyzed.   (Radford Dep. at p. 384).

As it turned out, the doses were higher. Hamilton, et al,

1987, reported the average thyroid dose to the Utirik Islanders

was 280 rads, nearly three times the upper range (95 rads)

reported in Conard. When confronted with this information at his

deposition, Radford testified he was "suspicious of reevaluations

of dose many years after the event," but admitted he did not know

the techniques or the basis for the reanalysis of the doses

undertaken by Hamilton. It is interesting that Hamilton is cited

as a reference for both Radford's original and supplemental

iodine reports, and yet Radford failed to either find or report

this reanalysis of doses. This does not reflect favorably upon

Radford's thoroughness and indeed suggests he was more concerned

in generating a particular result (association between low does

radiation and hypothyroidism), than how he arrived at that

result.

All that the plaintiffs offer in response is a footnote in

which they say "[t]hat a later reanalysis of the doses has

estimated them to be in the intermediate range does not reduce

the power of the causal association found at those ranges."

(Plaintiffs' Response Brief at p. 70, n. 110). This is a

concession that Conard does not support an association at low

ORDER RE SUMMARY JUDGMENT-   190

1   dose ranges. Essentially, the plaintiffs emphasize their belief

2   that Conard still proves radiation is "capable of causing"

3   subclinical or biochemical hypothyroidism.

4   Understandably, plaintiffs try to find support for Radford's

5   opinion elsewhere. Plaintiffs argue that Radford cites Kaplan

6   and Nagataki, two "low dose" studies, which support his finding

7   for irradiation induced hypothyroidism at low doses. Plaintiffs

8   also argue that Radford "explicitly" endorsed Dr. Mayer's

9   development of a dose-response relationship for hypothyroidism,

10   "demonstrating a causal association down to low doses."

11   Kaplan and Nagataki are both listed in the bibliography

12   attached to Radford's original iodine report. (Radford 1995

13   Iodine Rpt. at pp. 32-33). They are not included in the

14   bibliography attached to his supplemental report. (Supp. Rpt. at

15   pp. 9-10). Nowhere in his original report does Radford discuss

16   how Kaplan and Nagataki support his opinion of a causal

17   association between low dose radiation and hypothyroidism.

18   Radford premised his opinion on Conard and Simon and the

19   reliability of his methodology depends upon those studies.

20   Plaintiffs argue Dr. Ruttenber's analysis of the association

21   between radiation and chronic autoimmune thyroiditis, which

22   depends in part on Kaplan and Nagataki, applies equally to Dr.

23   Radford's analysis. <u>Daubert</u> is concerned with the methodology of

24   the expert who renders the opinion- in this case, Radford- and

25   not the methodology of any other expert. Secondly, all Ruttenber

26   opined in his report was that I-131 is "capable of causing"

27   autoimmune thyroiditis, not necessarily at low doses. Finally,

28   **ORDER RE SUMMARY JUDGMENT-   191**

1 | Radford is not clear in his reports whether he is discussing

2 | autoimmune hypothyroidism or non-autoimmune hypothyroidism

3 | (subclinical or clinical). If it is the former (autoimmune

4 | hypothyroidism), then Ruttenber's analysis may have something in

5 | common because chronic thyroiditis is also an autoimmune

6 | condition. Plaintiffs say this is the case because Ruttenber

7 | offered doubling doses for a different type of hypothyroidism

8 | (direct cell-killing non-autoimmune) and those doses (350

9 | external rads for biochemical hypothyroidism and 750 external

10 | rads for clinical hypothyroidism) are not inconsistent with a

11 | finding of a causal association between low dose radiation and

12 | **autoimmune hypothyroidism.**[132]

13 | Nowhere in his reports does Radford cite to Mayer as support

14 | for his (Radford's) opinion of a generic causal association

15 | between low dose radiation and hypothyroidism. At his

16 | deposition, Radford was asked about Mayer's dose-response curve

17 | for hypothyroidism. From various scientific articles, Mayer

18 | generated data points which were mapped onto a dose-response

19 | curve. Based on this curve, Mayer concluded the dose of I-131 at

20 | which the risk of hypothyroidism doubles is approximately 50

---

[132] The fact Radford relies on two studies, Simon and Conard, which evaluate thyroid hypofunction on the basis of TSH levels may suggest he was evaluating biochemical or subclinical hypothyroidism, but that does not necessarily mean it is **autoimmune** hypothyroidism. Ruttenber's non-autoimmune subclinical or biochemical hypothyroidism also depends on detection through TSH levels. (Ruttenber 1995 Iodine Rpt. at p. 13). Plaintiffs do not assert that Simon and Conard clearly deal with an autoimmune process as opposed to a non-autoimmune process.

ORDER RE SUMMARY JUDGMENT-   192

1 rads, with upper and lower bounds between 30 and 80 rads.[133]

2   Mayer's opinion is not reliable. Radford clearly recognized

3 the limitations of the underlying epidemiological studies upon

4 which Mayer relied for his dose-response curve. Radford

5 acknowledged he had not personally reviewed those studies to

6 determine whether the lines on Mayer's graph were properly

7 plotted. Although Radford reviewed some of the high dose

8 studies, he was "not so familiar" with the **low dose** studies.

9 Radford stated that Mayer's graph was "not a terribly good fit,

10 but it is the best fit you can do with the range of data that

11 exist." (Radford Dep. at p. 536).

12   Radford obviously did not give a ringing endorsement to

13 Mayer's work. Because of Radford's lack of familiarity with the

14 studies on which Mayer's graph is based, in particular the low

15 dose studies, the court fails to see how Radford could say his

16 opinion is supported by Mayer's work. Plaintiffs' counsel may

17 claim Mayer's work is supportive, but of course it is Radford

18 himself who must show how it is supportive of his methodology.

19   For all the reasons set forth above, the court finds

20 Radford's opinion of a causal association between **low dose**

21 radiation and hypothyroidism (autoimmune or non-autoimmune) is

22 not the product of a scientifically, methodologically reliable

23 analysis. Furthermore, Radford's failure to supply causative

24 risk estimates renders his opinion irrelevant to a jury's

25 ───────────────────

   [133] In his report, Mayer did not make a distinction between
26 autoimmune and non-autoimmune hypothyroidism, although
   plaintiffs' counsel subsequently asserted he was referring to
27 autoimmune hypothyroidism.

28
   ORDER RE SUMMARY JUDGMENT-   193

1 determination of whether radioiodine is a "more likely than not"

2 cause of an individual's hypothyroidism (whether that is

3 autoimmune hypothyroidism or non-autoimmune hypothyroidism).[134]

4

5 **(4) Parathyroid Adenomas and Hyperparathyroidism[135]**

6 In his 1995 iodine report, Radford offered the following

7 about these conditions:

8   Another medical consequence of radioactive iodine
        uptake into the thyroid gland is the production
9   of parathyroid adenomas accompanied by hyperpara-
        thyroidism (Rosen, et al., 1984). This problem
10   has been investigated among people given head
        and neck irradiation for medical purposes (Cohen,
11   et al., 1990), persons given radiation therapy
        for Hodgkin's disease (Hancock, et al., 1991),
12   as well as in the Adult Health Study population
        at Hiroshima and Nagasaki (Fujiwara, et al., 1992).
13   The frequency of this condition has been shown
        to be related to radiation exposure. The
14   sensitivity of production of this abnormality
        appears to be close to that found for thyroid
15   adenomas (Fujiwara, et al., 1992). In applying
        these findings to the situation where exposure has
16   been to [I-131] predominantly, calculations show
        (Finston Report in this case) that the beta and
17   gamma radiation from iodine would **likely** reach
        some of the parathyroid tissue adjacent to the
18   thyroid gland, **though with reduced doses from
        beta particles.** On this basis, therefore, para-
19   thyroid adenomas and consequent hyperparathyroidism
        may be related to iodine exposure arising from the
20   Hanford site.

21 _____

[134]   There is no reason to wait for the individual causation
22 stage as Radford claims is necessary. Neither Radford or anyone
    else can identify the specific clinical factors that a physician
23 could use to determine causation in the absence of causative risk
    estimates supported by properly conducted epidemiological
24 studies. The plaintiffs need epidemiological evidence in order
    to show that an agent is a "more likely than not" cause of a
25 disease (i.e. that there was a doubling of the risk from exposure
    to the agent).

26
[135]   Defendants dispute the compensability of nodules and
27 adenomas as physical injuries. That is discussed infra.

28
ORDER RE SUMMARY JUDGMENT-   194

1 (Radford 1995 Iodine Rpt. at p. 20)(Emphasis added).

2   Radford went on to say:

3   Parathyroid adenomas have radiation-related
            risk co-efficients that are **somewhat less**
4   than those for thyroid cancers or adenomas,
            and also fit the linear, no threshold dose
5   response relationship (Fujiwara, <u>et al.</u>, 1992).
            The age-dependence for induction by radiation
6   is also present for this disease (Fujiwara, <u>et</u>
            <u>al.</u>, 1992), similar to that for thyroid adenomas.
7   Since incidence of parathyroid adenomas and hyper-
            parathyroidism is **rare**, these cases can be
8   separately evaluated on an individual basis from
            exposure data with regard to radiation-related
9   causation.

10 (<u>Id</u>. at p. 28) (Emphasis added).

11   Defendants contend Radford has no idea whether I-131 from

12 Hanford emissions could have cause parathyroid adenomas and

13 hyperparathyroidism. They point out Radford's statement that

14 "parathyroid adenomas and consequent hyperparathyroidism **may** be

15 related to iodine exposure arising from the Hanford site." They

16 also note he does not provide any risk estimates, instead

17 proposing to evaluate claims "on an individual basis from

18 exposure data with regard to radiation-related causation."

19 According to defendants, Radford does not explain how the claims

20 should be analyzed or how, in the absence of epidemiological

21 proof, causation could be established at the **low doses** alleged.

22 Therefore, defendants argue Radford's opinion is irrelevant and

23 should be excluded under Prong 2 (fit/relevancy) of <u>Daubert</u>.

24   Plaintiffs point out that defendants do not challenge any of

25 the studies Radford cites in support of his "position." While it

26 is true defendants do not discuss any of these studies, the more

27 significant question is what exactly is Radford's "position."

28 **ORDER RE SUMMARY JUDGMENT-   195**

1 Based on the studies cited by him (Cohen, Hancock and Fujiwara),

2 Radford asserts the frequency of parathyroid adenomas and

3 hyperparathyroidism has been shown to be "related" to radiation

4 exposure. This is the equivalent of saying radiation is "capable

5 of causing" these conditions. Radford does not say anything

6 about the dose levels, although they may be high considering the

7 studies appear to involve doses administered for medical and

8 therapeutic purposes.[136] However, Radford then follows up by

9 saying where exposure has been to I-131 predominantly (which is

10 the situation with the plaintiffs), it is "likely" that beta and

11 gamma radiation from iodine would reach some of the parathyroid

12 tissue adjacent to the thyroid gland, though with "reduced" doses

13 from beta particles. He concludes that parathyroid adenomas and

14 hyperparathyroidism "may" be related to iodine exposure arising

15 from the Hanford site. This is a very tentative opinion that low

16 doses of I-131 which finally reach the parathyroid gland are

17 "capable of causing" parathyroid adenomas and

18 hyperparathyroidism.

19   The defendants appear to challenge Radford only on the basis

20 of fit/relevancy. Radford has not supplied causative risk

21 estimates, and indeed it is not apparent how he could do so based

22 on his conclusion that parathyroid adenomas and

23 hyperparathyroidism at best "may" be related to iodine exposure

24 arising from the Hanford site. Therefore, his opinion is

25 irrelevant to a jury determination of whether radioiodine is a

26 _____

[136] We know for a fact the Hancock study involved high

27 doses. See discussion supra re Graves' disease.

28 **ORDER RE SUMMARY JUDGMENT-   196**

1 "more likely than not" cause of any individual's parathyroid

2 adenomas or hyperparathyroidism. For that reason, Radford's

3 opinion regarding parathyroid adenomas and hyperparathyroidism

4 will be stricken.

5

6 ### d. Conclusion

7 The court will grant defendants' motion in limine insofar as

8 Radford's opinions about non-neoplastic conditions, and his

9 opinion about application of an individual susceptibility factor

10 to increase thyroid cancer risk estimates.

11 The court will deny defendants' motion in limine insofar as

12 Radford's opinion about equal effectiveness between I-131 and

13 external radiation, and the age-related adjustments to his

14 thyroid cancer risk estimates for the 0 to 4 and 5 to 9 age

15 groups.

16

17 ### 4.   Viktor Ivanov

18 ### a.   Introduction

19 Dr. Ivanov is the author of an expert report dated October

20 1995. Ivanov is the deputy director of the Russian Federation

21 Medical Radiological Research Center (MRRC) and the director of

22 the World Health Organization (WHO) Collaborating Center for

23 Radiation Epidemiology at Obninsk, Russia. Ivanov has been

24 involved in studies of the health effects of the 1986 Chernobyl

25 accident. His affidavit (Foulds Ex. 61) states his work in

26 radiation epidemiology began in 1986 after the Chernobyl accident

27 and has continued since that time.

28 **ORDER RE SUMMARY JUDGMENT-   197**

1   Ivanov's report discusses the Chernobyl data pertaining to

2   two oblasts "which had relatively low dose levels." These are

3   Kaluga Oblast, located approximately 600 kilometers northwest of

4   Chernobyl, and the Bryansk Oblast adjoining the Kaluga Oblast to

5   the south. Ivanov's discussion is limited to data from these

6   areas because they involve "areas and groups of lower dose

7   exposures that may be comparable to doses experienced by

8   residents downwind from Hanford, Washington, U.S.A." (Ivanov

9   Rpt. at p. 1).

10   Ivanov refers to a case-control study in the Bryansk region

11   revealing the "index of relative risk of thyroid cancer is 7.15

12   when radiation [dose] is 1 Gy [1 Gray or 100 rads]." According

13   to Ivanov, this means that 88% of detected cancer cases are due

14   to radiation exposure. (Id. at p. 5). Ivanov also refers to

15   finding a "relationship between dose and non cancer thyroid

16   diseases (radiation risks)" as an "unexpected result" of an

17   investigation related to the "Kaluga cohort." Ivanov indicates

18   that "[e]uthyroid goiter makes the main contribution (to 80%) to

19   the structure of non cancer diseases." (Id.)

20   Defendants challenge the reliability of the risk estimates

21   derived from these two studies- the thyroid cancer case control

22   study from Bryansk and the non-cancerous thyroid disease study

23   from Kaluga- because of what they claim is the preliminary nature

24   of the studies.

25

26   **b.   Thyroid Cancer Case-Control Study**

27   This study was based on seventeen (17) thyroid cancer cases

28   **ORDER RE SUMMARY JUDGMENT-   198**

1   and matching controls (persons without thyroid cancer).[137]   In

2   his affidavit, Ivanov says there were 107 controls.   Because

3   there were only 17 cases, Ivanov acknowledges his risk estimates

4   are preliminary. (Ivanov Dep. at pp. 110-11).   Those estimates

5   are as follows based on three separate dose categories for a

6   group described as "children and adolescents of Bryansk oblast:"

7   1) 5-60 rads- relative risk 0.46 with a confidence interval range

8   between 0 and slightly more than 2.0; 2) 60-140 rads- relative

9   risk of 7.15 with a confidence interval range between 1.8 and

10   38.9; and 3) over 140 rads- relative risk of 7.15 with a

11   confidence interval range between 1.0 and 60.2.   (Figure 5

12   attached to Ivanov Rpt.; Ivanov Dep. at pp. 84-87).

13   The defendants begin their critique of Ivanov's case-control

14   study by pointing out that Ivanov never took any academic courses

15   in **cancer** epidemiology and the Bryansk study represents the first

16   case-control study he has ever personally conducted.   They also

17   note that prior to 1986, Ivanov was not involved with cohort

18   studies, other than what he refers to as life-span cohort

19   studies.[138]   (Ivanov Dep. at pp. 8-9; 43; 239).   Nonetheless,

20   defendants say they do not dispute Ivanov's qualifications.   That

21   being the case, the critical question is whether Ivanov's

---

[137]   A case-control study is one that starts with
identification of persons with the disease and a suitable control
group of persons without the disease.   The researcher compares
past exposures.   If a past exposure is associated with or caused
a disease, the researcher expects to find a higher proportion of
past exposure among the cases (versus the controls).   "Reference
Guide on Epidemiology" at p. 136.

[138]   The Kaluga cohort study, at issue here, apparently was
not a life-span study.

ORDER RE SUMMARY JUDGMENT-   199

1 purported lack of experience somehow manifests itself by way of

2 specific methodological deficiencies in his case-control study

3 (or his cohort study, discussed infra).

4   Secondly, defendants contend that though the **results** of the

5 case-control study have been presented at several international

6 conferences, the **study** itself is available only in Russian and

7 not in a form which can be critically reviewed by international

8 scientific organizations such as the UNSCEAR and BEIR committees.

9 The study has not been published in a peer-reviewed scientific

10 journal.[139] According to defendants, to the extent there is a

11 protocol for the study, it also is available only in Russian and

12 has not been submitted to any scientists outside of Russia for

13 review or comment. Defendants assert the lack of a comprehensive

14 protocol comporting with international scientific standards and

15 setting forth clear and uniformly applied criteria has led the

16 "broader scientific community" to discount the data generated by

17 Ivanov. Specifically, they cite the comments of a Dr. Williams

18 at the April 1996 Vienna Conference that Chernobyl thyroid cancer

19 data was not "deliberately ignored," but "only data from those

20 countries which have agreed to abide by internationally accepted

21

22

23 ———————————————

       [139] Plaintiffs do not dispute that the study has not been
24 published. Instead, they argue Ivanov has published a number of
    "basic papers" regarding the data from Russia, Ukraine and
25 Belarus which confirm the "preliminary estimates" of the study.
    (Ivanov Affidavit at p. 4). Likewise, plaintiffs contend
26 Ivanov's **"data"** has been published, cited and subjected to
    worldwide scrutiny. However, that does not mean the study has
27 been published.
28
    ORDER RE SUMMARY JUDGMENT-   200

1 uniform diagnosis standards were presented."   (Vienna

2 Conference[140] at p. 235).

3   Plaintiffs contend Ivanov's data was gathered pursuant to

4 "internationally approved" WHO protocols.   In his affidavit,

5 Ivanov states that "[s]ince the basic primary documents (such as

6 questionnaires) were developed in cooperation with an

7 international team of experts, using internationally accepted

8 protocols, we felt it was not necessary to submit duplicate

9 protocols for the study."   (Affidavit at p. 3).

10   Defendants correctly observe that Ivanov does not refer to

11 any WHO protocols in his affidavit.   Nor is there any dispute

12 about Ivanov's deposition testimony that the case-control study

13 was not submitted to international experts for review and

14 comment.   (Ivanov Dep. at pp. 94-95).   Defendants contend the WHO

15 protocols are general protocols which do not provide the details

16 necessary for individual studies.   They note the WHO protocols

17 specify that comparisons be made on the basis of gender and age.

18 Ivanov's case control study does not contain a gender breakdown

19 and there are no specific age categories.[141]

20   Although the view of the international scientific community

21 about the Chernobyl data **in general** is undoubtedly pertinent, the

22 defendants do not suggest on that basis alone, Ivanov's **specific**

23
_____

24   [140] IAEA, "One Decade After Chernobyl: Summing up the
   Consequences of the Accident, Proceedings of an International
25 Conference," Vienna (1996).   Defendants' Ex. 123.

26   [141] Dr. Radford's testified that age and sex are the major
   modifiers of risk for thyroid cancer.   Plaintiffs' expert, Dr.
27 Clifton, concurs.

28 **ORDER RE SUMMARY JUDGMENT-   201**

1 case-control study is per se unreliable. Although it is

2 certainly relevant that the study has not been published in a

3 peer-reviewed journal and has not been available for

4 international review, that alone is insufficient to exclude

5 Ivanov's opinion. Peer review and general acceptance are but two

6 factors for consideration.

7 At his deposition, Ivanov acknowledged the 17 thyroid cancer

8 subjects did not have direct thyroid measurements. Rather, the

9 doses for those subjects (as well as for the controls) were

10 reconstructed. (Ivanov Dep. at pp. 124-25). The dose

11 reconstruction was based on a ratio between Cesium-137

12 contamination and other environmental measures of Iodine-131

13 contamination. (Id. at pp. 132-33). Ivanov admitted the

14 presumed ratio was not valid for the purpose of reconstructing I-

15 131 doses:

16   And the main conclusion that for iodine
        dosimetric purpose, you should use iodine
17   contaminated information, **not cesium. Very
        wrong to use cesium information for iodine.**
18   But unfortunately, we have not such type of
        approach.
19

20 (Id. at p. 140).

21 Plaintiffs acknowledge the doses received by the 17 thyroid

    cancer subjects were not directly measured, but contend they were
22
    "properly reconstructed." However, they do not claim Ivanov was
23
    mistaken in his deposition testimony or that his testimony has
24
    somehow been misinterpreted. Therefore, the fact other studies
25
    used Cesium-137 for dose reconstruction purposes (as cited by
26
    plaintiffs) is not significant. It does not prove the use of
27

28 **ORDER RE SUMMARY JUDGMENT-   202**

1 Cesium-137 is reliable for reconstructing 1-131 doses to the

2 thyroid.[142]

3   Next, defendants argue the case-control study risk estimates

4 are unreliable because they fail to take into account certain

5 confounding factors, including iodine deficiency in the Bryansk

6 region and intensive medical screening of the Bryansk population.

7 Ivanov acknowledges the Bryansk oblast is considered an "endemic

8 goiter" region (iodine deficient due to inadequate dietary

9 intake) and that "it will be good to clarify this, the influence,

10 the role of endemic in excessive or increasing cancer and non-

11 cancer disease." (Ivanov Dep. at pp. 163-164). This is a factor

12 which needs to be considered because individuals who are iodine

13 deficient absorb a greater amount of radioiodine and also produce

14 more thyroid-stimulating hormone (TSH). (Id. at pp. 169-70).

15   As Ivanov indicates in his report, the children in the

16 Bryansk and Kaluga regions receive regular medical examinations

17 and laboratory testing. (Ivanov Rpt. at p. 2). Ivanov

18 acknowledges that "increased medical screening" is a possible

19 explanation for the increased incidence of thyroid cancer. Such

20 screening leads to an increased detection of cases compared to

21 the unscreened, unexposed population by a factor between 2 and 3.

22 (Ivanov Dep. at pp. 160-63).

23   The plaintiffs contend iodine deficiency and overscreening

24 _____

25 [142] Plaintiffs cite work of a Valerie Beral and Warren
   Sinclair which they claim shows the accuracy of the doses from
   Chernobyl. (Plaintiffs' Response Brief at p. 15). However,
26 there is no indication this work pertains specifically to
   Ivanov's dosimetry or to the reliability of using cesium to
27 calculate iodine dose.

28

ORDER RE SUMMARY JUDGMENT-   203

1 | are highly individual factors which can only be assessed on a

2 | case by case basis. The court is not persuaded. These factors

3 | affect the validity of the study and the extent to which any

4 | observed association is causal at a population level. Until

5 | causation is reasonably established at that level, there is no

6 | issue to be addressed at an individual level. Ivanov says as

7 | much in his affidavit:

8 | It is a well known fact that other considerations
which might possibly have affected individual
9 | radiation risks would include iodine deficiency
(stemming from goitrogenic areas)- genetic
10 | predisposition, and to some extent, the comprehensive
medical screening. . . . **Quantity estimates of the**
11 | **contribution of other confounding factors derived
from the Chernobyl data [have] not been published yet,**
12 | **<u>which is why it is impossible to attribute registered</u>
<u>increase in thyroid cancer among those children in</u>**
13 | **<u>1986 to above identified confounding factors rather</u>
<u>than I-131 exposure to the thyroid.</u>**

14 | (Ivanov Affidavit at p. 4) (Emphasis added).

15 | Finally, defendants say another problem with Ivanov's case-

16 | control study is that because it is based on so few cases (17

17 | cases), it does not have sufficient statistical power to generate

18 | reliable dose estimates. The results are not statistically

19 | significant for two of the three dose categories (5-60 rads and

20 | over 140 rads) because the confidence interval includes 1.0- the

21 | background incidence of thyroid cancer. With regard to the 60-

22 | 140 rads category, there is statistical significance. However,

23 | defendants observe that the confidence interval is quite broad

24 | (1.8 to 38.9). According to defendants, statistical significance

25 | was reached in this category only because the study ignored

26 | differences in gender and has broad age and dose categories.

27
28 | **ORDER RE SUMMARY JUDGMENT-   204**

1   At his deposition, Ivanov testified the age category

2 referred to is children 0 to 14 and teenagers from 15 to 17.

3 There was no distinction on the basis of sex, although Ivanov

4 readily acknowledges the scientific literature indicates women

5 are at a higher risk for thyroid cancer than men. Ivanov

6 conceded that had he presented risk estimates based on men and

7 women separately, a statistically significant result would not

8 have been produced. Nor would such a result have been produced

9 if he had used narrower age categories. (Ivanov Dep. at pp. 239-

10 40).

11   Defendants assert Ivanov's data is too limited to permit

12 evaluation of a dose-response relationship. With regard to

13 thyroid cancer, a linear dose-response relationship is expected-

14 the more intense the exposure, the greater the risk of

15 disease.[143]   (Ivanov Dep. at pp. 76-77). According to

16 defendants, Ivanov could not test for such a relationship because

17 of the limited number of cases in his study. As defendants point

18 out, the lack of statistically significant results in the 5-60

19 rads category and the over 140 rads category shows a lack of

20 dose-response relationship. There is not a greater risk of

21 disease with greater exposure (exposure over 140 rads). Because

22 of the lack of a statistically significant result in the 5-60

23 rads category, it cannot be determined whether the result in the

24 60-140 rads category represents a true dose-response

25 relationship.

26 _____

    [143]   The absence of dose-response relationship in **thyroid**

27 **cancer** studies is significant for this reason.

28 **ORDER RE SUMMARY JUDGMENT-   205**

1   The plaintiffs offer a weak response. They ignore the lack

2   of statistical significance in the 5 to 60 rads category and

3   simply point out that the upper limit of the confidence interval

4   exceeds 2.0.[144] They also assert that Ivanov is engaged in an

5   "ongoing" study, many additional cases will be added, and "[t]his

6   will increase the association." According to plaintiffs, "Ivanov

7   provides very preliminary dose response/risk relationships and,

8   with improved dosimetry and additional data [,] dose response

9   risk ranges will be generated with greater association."

10   Ivanov is refreshingly candid. He readily acknowledges the

11   preliminary nature of his risk estimates and the need to consider

12   factors affecting those estimates. He is definitely proposing to

13   testify about matters growing naturally and directly out of

14   research he has conducted independent of this litigation. The

15   Ninth Circuit opined that this is the most persuasive basis for

16   concluding the expert's opinion is derived by the scientific

17   method. On the other hand, Ivanov's case-control study has not

18   been published and has not been subjected to scientific scrutiny

19   through peer review. And as noted above, there has not been

20   _____

        [144] According to plaintiffs, statistically significant
21   results within an arbitrary 95% confidence level could not be
     provided "without having to group the cohort by ages 0-14 and 15-
22   17 which most likely underestimated risks." Plaintiffs cite
     Ivanov's deposition testimony in support of this, but the court
23   fails to see where he says any such thing.
        Actually, it seems that by lumping the sexes together, the
24   result might be to underestimate the risk for girls, but at the
     same time overestimate the risk for boys. Ivanov acknowledged
25   the risk is greater for girls. Similarly, by lumping ages
     together, the result might be to underestimate the risk for very
26   young children, while overestimating the risk for the teenagers.
     The general consensus is that risk for thyroid cancer diminishes
27   with increased age.
28   **ORDER RE SUMMARY JUDGMENT-   206**

1 "general acceptance" of his risk estimates within the scientific

2 community.

3   While Ivanov may have employed a sound scientific

4 methodology to produce his **preliminary results**, the point is that

5 the results are **preliminary**. While preliminary results may

6 ultimately turn out to be reliable, the point is we do not know

7 for sure if that will be the case. There are a number of factors

8 which could reasonably affect the validity of the risk estimates,

9 particularly the estimate for the 60 to 140 rads category.

10 Consideration of those factors (small sample size, confounding

11 variables, lack of statistical robustness) may well make the

12 results for that category statistically insignificant (i.e. widen

13 the confidence interval such that 1.0 is included therein).[145]

14   Statistical significance is vital here. The issue is not

15 _____

16   [145] Once an association has been found between exposure to a
substance and a disease (in this case, the 7.15 relative risk in
the 60 to 140 rads category), researchers must still consider

17 whether the association reflects a true cause-effect relationship
or a spurious finding. They first look for alternative

18 explanations for the association, such as bias or **confounding
factors**. The exposure and the disease may be caused by a

19 confounding factor. A confounding factor is both a risk factor
for the disease and associated with the exposure of interest. To

20 identify potential confounding factors, the researcher must
assess a range of factors that could influence risk. This

21 procedure often involves complex statistical manipulations
comparing the overall risk of exposure with the risk when

22 identified potential confounding factors have been removed from
the calculation. Stratification is one of the techniques used to

23 **control** for confounding factors during data analysis. It reduces
or eliminates confounding by evaluating the effect of exposure at

24 different levels (strata) of exposure of the confounding
variable. Statistical methods can then be applied to combine the

25 different results of each stratum into an overall single estimate
of risk. "Reference Guide on Epidemiology" at pp. 157-60.

26   In this case, defendants suggest Ivanov should have
"stratified" his data on the basis of gender and more tightly

27 drawn age categories.

28
ORDER RE SUMMARY JUDGMENT-   207

1 simply whether I-131 is "capable of causing" thyroid cancer.[146]

2 That is already a given. Ivanov's results purport to show a

3 doubling of the risk for children and adolescents at 60 rads.

4 The risk, as reported, is statistically significant because the

5 low end of the range (1.8 to 38.9) exceeds 1.0 (the background

6 rate).[147]

7   The statistically significant result Ivanov reported for the

8 60 to 140 rads category is simply too unreliable for the reasons

9 cited: uncertainty in dose estimates, confounding variables

10 (iodine deficiency, overscreening, age, sex), lack of dose-

11 response, etc. The lack of scientific scrutiny of Ivanov's

12 specific results[148] reinforces the conclusion that they are

13 simply too uncertain and too unreliable to raise an inference of

14 _____

15 [146] One should compare Ruttenber's opinion about chronic
thyroiditis. Ruttenber did not concern himself with doubling of
16 risk. The lack of statistical significance in one of the studies
cited by Ruttenber (Kaplan, et al.) in support of his opinion
17 that radiation is "capable of causing" chronic thyroiditis is not
enough to render that opinion unreliable. Here, on the other
18 hand, Ivanov's results stand alone and are at least potentially
offered for the proposition that exposure to 60 rads or more is a
19 "more likely than not" cause of an individual's thyroid cancer.

20 [147] It is not completely clear whether Ivanov's results are
based on a 95% confidence interval, although at his deposition,
21 Ivanov said he tries to use 95% "as a rule." (Ivanov Dep. at p.
75).
22
     [148] Plaintiffs cite a number of other studies (Sobolev, Ron
23 and Demidchik) which they assert compare favorably to and confirm
Ivanov's estimates. However, the concern here is with how **Ivanov**
24 derived his results. Only Ivanov's estimates are at issue. It
is only those estimates which the plaintiffs use as support for
25 their thyroid cancer claims. Furthermore, there is no indication
of how similar these other studies are to Ivanov's study and
26 whether they compensated for all of the shortcomings of the
Ivanov study in terms of accuracy of dose estimates,
27 consideration of confounding variables, etc.
28
ORDER RE SUMMARY JUDGMENT-   208

1 a doubling of the risk for children and adolescents at 60

2 rads.[149]

3   The risk estimates Ivanov derives from his thyroid cancer

4 case-control study are inadmissible under both prongs of <u>Daubert</u>-

5 reliability and relevancy. Ivanov's one statistically

6 significant result for the 60-140 rads category is so unreliable

7 that its relative risk estimate (7.15) cannot be used to meet the

8 "doubling of risk" ("more likely than not") standard. His

9 estimates for the 5-60 rads category and the over 140 rads

10 category are not statistically significant because they include

11 1.0 in their confidence intervals. 1.0 is the same as the

12 background risk and therefore, "doubling of risk" obviously

13 cannot be proven.[150]

14

15   **c.   Non-Cancerous Thyroid Disease Study**

16   This is a cohort study based on medical examinations of

17 6,000 children and teenagers in the Kaluga Oblast.[151] Ivanov's

18 _____

19   [149] Ivanov's report and his chart say nothing about risk
   estimates for adults.

20
   [150] If plaintiffs are arguing that all Ivanov's report
21 stands for is that I-131 is "capable of causing" thyroid cancer
   at 20 rads or less in children and adolescents, obviously that is
22 insufficient to meet the "doubling of risk" standard.

23   [151] In cohort studies, the researcher identifies two groups
   of individuals: 1) individuals who have been exposed to a
24 substance that is thought might cause the disease; and 2)
   individuals who have not been exposed. Both groups are followed
25 for a specified length of time, and the proportion of each group
   that develops the disease is compared. If the exposure is
26 associated with or causes the disease, the researcher would
   expect a greater proportion of the exposed individuals to develop
27 the disease. "Reference Guide on Epidemiology" at p. 134.

28 **ORDER RE SUMMARY JUDGMENT-   209**

report says nothing about risk estimates derived from this study.

However, it turns out that Ivanov did prepare such estimates.

These estimates were included in a table which was part of a

presentation made by a Dr. Tsyb at the April 1996 Vienna

Conference. The table is entitled "Comparison of Radiation Risk

Coefficients for Non-Cancer Thyroid Diseases in Children and

Adolescents of Kaluga Cohort and Atomic Bomb Survivor Cohort."

In that table, Ivanov concludes the excess relative risk for non-

cancerous thyroid disease per 1 gray (100 rads) is 0.2. The

confidence interval range is 0.06 to 0.34. Ivanov testified he

considered this to be a reliable risk estimate with respect to

non-cancerous thyroid disease. (Ivanov Dep. at pp. 178-79).

According to defendants, **if Ivanov's data is reliable**, it

show the risk of non-cancer thyroid conditions increases 20% (0.2

over the baseline risk of 1.0) for each 100 rads of exposure.

Therefore, a dose of 500 rads is necessary to double the risk of

these conditions (20% x 5 = 100%; 100 rad x 5 = 500 rads).

Defendants claim, however, that Ivanov's data is unreliable and

therefore, so are his causative risk estimates.

Defendants assert one problem is the study achieved

significance only by lumping together several different thyroid

conditions. Euthyroid goiter[152] made up 80% of the non-cancer

thyroid disease. (Ivanov Rpt. at 5; Ivanov Dep. at pp. 214-15).

The other 20% included autoimmune thyroiditis and nodules. (Id.)

Thus, say defendants, Ivanov's study fails the "specificity"

_____

[152] Goiter in a normally functioning thyroid.

ORDER RE SUMMARY JUDGMENT-   210

1 criterion.

2 The court fails to see where plaintiffs respond to this

3 particular criticism. The "specificity" criterion[153] is met

4 where there is a lumping of similar types of diseases (i.e.

5 autoimmune diseases sharing a similar autoimmune mechanism).

6 Ivanov goes further than this. "Euthyroid Goiter" is an

7 enlargement of the thyroid gland due to diminished thyroid

8 hormone production, but without clinical hypothyroidism. Merck

9 Manual, Sixteenth Ed. (1992) at p. 1084. Plaintiffs do not

10 assert it is an autoimmune condition like autoimmune thyroiditis.

11 Thus, Ivanov's non-cancerous thyroid condition is extra broad and

12 consequently limits the inference that can be raised about the

13 relationship between radiation and a particular thyroid disease.

14 Defendants refer to deposition testimony from Ruttenber that

15 an analysis combining different diseases is difficult to

16 interpret and that grouping of thyroid diseases is not a useful

17 thing in determining the relationship between exposure and

18 disease. (Ruttenber Dep. at p. 172). Ruttenber made this

19 comment in regard to results published in Wong, et al. 1993.[154]

20 Ivanov compared his risk estimates for non-cancer thyroid disease

21 to estimates reported in Wong for the broad category of "Thyroid

22 Disease." Wong acknowledged that due to the overlap in thyroid

23 _____

24 [153] An association exhibits "specificity" if the exposure is
associated only with a single disease or type of disease.
25 "Reference Guide on Epidemiology" at p. 163.

26 [154] Wong, et al., "Noncancer Disease Incidence in the Atomic
Bomb Survivors: 1958-1986," **Radiation Research**, Vol. 135, No. 3
27 (1993). Defendants' Ex. 129.

28 **ORDER RE SUMMARY JUDGMENT-   211**

diagnoses, "the effects of ionizing radiation on a specific thyroid disorder" could not be delineated in his study.   (Wong at p. 425).

Another problem, contend defendants, is the fact 80% of the non-cancer thyroid disease identified is goiter.   Kaluga, like Bryansk, is an endemic region in that individuals suffer from iodine deficiency due to dietary intake.   (Ivanov Dep. at p. 180).   This is a confounding factor which increases the risk for thyroid disease.

At his deposition, Ivanov could not identify any "non-Russian" study or publication concluding that I-131 causes goiter.   He referred to a Russian publication, but he did not discuss the results thereof and stated somewhat cryptically that "it's not enough epidemiological background for this."   Ivanov identified the Wong study as the only "good" international epidemiological investigation for non-cancer thyroid disease, and asserted its risk estimates were similar to his own.   However, he admitted he was not aware of any epidemiological publication, including Wong, dealing specifically with goiter.   (Ivanov Dep. at pp. 216-17).   Defendants contend the fact 80% of the disease identified in the study was euthyroid goiter is an "anomalous" result that does not satisfy the consistency criterion which epidemiologists use to assess causation.[155]

In his affidavit, Ivanov states "[i]t is not known if

_____

[155]   The court assumes defendants mean "consistency" in the sense that there are no other studies reporting euthyroid goiter as such a high percentage of the total amount of non-cancerous thyroid disease.

ORDER RE SUMMARY JUDGMENT-   212

radiation risks might be related to iodine deficiency." (Ivanov

Affidavit at p. 5). However, this is merely a recognition that

there is a risk which should be taken into account. Plaintiffs

admit euthyroid goiter "may be due to a variety of causes." They

assert there are studies which "explore goiter as a possible

precursor to thyroid cancer." However, plaintiffs do not

identify any study which says iodine-131 is capable of causing

goiter. Therefore, it is irrelevant whether goiter can develop

into thyroid cancer.

According to defendants, a third problem with Ivanov's

cohort study is that it is based on subjective clinical diagnosis

and not the results of objective medical testing, such as

ultrasound examination and hormone measurements. Furthermore,

defendants cite deposition testimony from Ivanov in which he

admits the results of the objective testing, when analyzed

separately, did not show any excess compared with the controls.

Ivanov was shown a table from the 1996 scientific report by the

World Health Organization (WHO). For each category listed on the

table- cysts, nodules and autoimmune thyroiditis- Ivanov

acknowledged the control group (the unexposed group) had a higher

incidence of such conditions.[156] (Ivanov Dep. at p. 185).

Defendants also assert that hormone measurements failed to

show any "dose-response relationship."[157] However, the hormone

---

[156] The results were based on ultrasonic thyroid gland investigations. (Ivanov Dep. at p. 184).

[157] Presumably defendants mean the measurements failed to show an increase in the level of thyroid hormone with increased exposure.

ORDER RE SUMMARY JUDGMENT-   213

1   measurements to which defendants refer (and on which they

2   questioned Ivanov) refer to children in Bryansk, not Kaluga.

3   (Ivanov Dep. at pp. 186-87). Secondly, Ivanov would not admit

4   that the hormone measurements failed to show a dose-response

5   relationship. Ivanov asserted the measurements were taken of

6   children from relatively equal radiocontaminated areas and there

7   was no comparison between "clean" and contaminated areas. (Id.

8   at 188-89).

9   The plaintiffs have consistently asserted the absence of a

10   dose-response relationship is not significant with regard to

11   autoimmune thyroid disease. A dose-response relationship assumes

12   the more intense the exposure, the greater the risk of disease.

13   However, a dose-response relationship may not be observed when

14   there is a threshold phenomenon (i.e. low dose exposure may not

15   cause disease until the exposure exceeds a certain dose).

16   "Reference Guide on Epidemiology" at p. 164. This is precisely

17   what the plaintiffs assert is the situation with autoimmune

18   thyroid disease. The absence of a dose-response relationship is

19   not alone sufficient to discredit Ruttenber's opinion regarding

20   chronic thyroiditis and Radford's opinion about non-neoplastic

21   diseases. The court finds no compelling reason to treat Ivanov

22   differently in this regard.

23   Ultimately, it appears the plaintiffs are really not

24   concerned about supporting the risk estimate Ivanov reported for

25   his non-cancer thyroid study (excess relative risk per 1 gray

26   (100 rads) is 0.2 with confidence interval range of 0.06 to

27   0.34). Indeed, it appears they and Ivanov are willing to concede

28   **ORDER RE SUMMARY JUDGMENT-   214**

1 this risk estimate is so preliminary as to be unreliable (and in

2 turn, that it is improper for defendants to derive a doubling

3 dose of 500 rads from that risk estimate). According to Ivanov:

4   We first assessed coefficients of radiation
            risk for the noncancerous thyroid diseases. Even
5   though we did not manage to assess radiation risks
            for a **specific** noncancerous thyroid disease the results
6   are quite clear from an epidemiological point of view.
            It is evident that radiation risks cannot be assessed
7   **based on a small sample of the population.**

8 (Ivanov Affidavit at p. 5) (Emphasis added).

9   Plaintiffs argue that what is important from Ivanov's

10 results is the "imminent comparability of the Kaluga (est. dose

11 .2 Gy [20 rads]) and Bryansk oblast to Hanford, and therefore the

12 importance of this data to the trier of fact in establishing

13 elements of **general causation** . . . ." Of course, what

14 plaintiffs mean by "general causation" is the capability of I-131

15 to cause the diseases in question (not whether it is a "more

16 likely than not" cause of those diseases).[158] A relative risk

17 above 2.0 is not critical for proving I-131 is "capable of

18 causing" a disease.

19   For the various reasons cited above (i.e. lumping of

20 conditions, confounding factors etc.), Ivanov's data is simply

21 not reliable enough to derive any risk estimates for the Kaluga

22

23   [158] In his affidavit, Ivanov says the amount of I-131
released from Chernobyl far exceeds that released from Hanford,
24 although "thyroid dose to residents of Kaluga oblast **(about .2Gy)**
are comparable with those in Hanford." (Ivanov Affidavit at p.
25 5). The court assumes plaintiffs would argue this shows that I-
131 doses as low as 20 rads are "capable of causing" non-
26 cancerous thyroid disease and the Hanford population was exposed
to such doses, thus meeting what plaintiffs assert is their
27 generic causation burden.

28
ORDER RE SUMMARY JUDGMENT-   **215**

1 cohort with regard to non-cancerous thyroid disease in general.

2 The plaintiffs and Ivanov apparently are willing to concede as

3 much.   If all Ivanov is opining is that I-131 is "capable of

4 causing" non-cancerous thyroid disease, that will never be enough

5 to sustain a jury verdict based on the "more likely than not"

6 evidentiary standard.   Such an opinion does not "fit" and is not

7 relevant to plaintiffs' ultimate burden of proof.   Thus, Ivanov's

8 opinion must be stricken on the basis of Prong 2 of Daubert

9 (fit/relevancy).[159]

10   Striking Ivanov's opinion on the basis of "fit" makes it

11 unnecessary to further assess the reliability of his methodology.

12 Nonetheless, the question then becomes whether Ivanov's analysis

13 is even scientifically reliable enough to support the proposition

14 that I-131 is "capable of causing" non-cancerous thyroid disease,

15 in particular goiter, autoimmune thyroiditis, and nodules.

16   Goiter is a problem for reasons enunciated above.   Kaluga is

17 an iodine deficient area and there is no epidemiological study

18 specifically dealing with goiter which concludes I-131 is

19 "capable of causing" that condition.   Ivanov did not isolate

20 goiter in his study.   He lumped it together with other conditions

21 which limits the ability to infer a causal relation between

22 radiation and goiter specifically.   Consequently, the court finds

23 Ivanov's methodology is not even reliable for the proposition

24 _____

     [159] Ivanov's risk estimates for thyroid cancer are
25 unreliable. As such, all that is effectively left is an opinion
     that I-131 is "capable of causing" thyroid cancer. There is no
26 dispute about that. However, such an opinion alone cannot
     sustain a jury verdict based on the "more likely than not"
27 standard.

28
     ORDER RE SUMMARY JUDGMENT-   216

1 that I-131 is "capable of causing" goiter.

2   The court is also not convinced Ivanov has provided a

3 scientifically reliable opinion that I-131 is "capable of

4 causing" autoimmune thyroiditis, particularly in light of the

5 results of the objective testing which showed the control group

6 actually had a higher incidence of the disease as compared to the

7 exposed group. Plaintiffs cite the 1996 IPHECA (International

8 Programme on the Health Effects of the Chernobyl Accident) report

9 which found an increased level of anti-thyroid antibodies in

10 exposed children versus unexposed children. However, that still

11 does not take away from the fact Ivanov reported a higher

12 incidence of autoimmune thyroiditis in his control group versus

13 his exposed group.

14   The objective testing also showed a higher incidence of

15 nodules in the control group. However, nodules are a precursor

16 to thyroid cancer and there is no dispute I-131 is "capable of

17 causing" thyroid cancer. Therefore, the court does not believe

18 there is a serious dispute that I-131 exposure causes thyroid

19 nodules at the same dose levels as thyroid cancer (and therefore,

20 that the same doubling doses should apply). Indeed, the only

21 argument defendants raise about nodules is their compensability

22 as physical injuries which is discussed infra.

23

24   d.   Conclusion

25   For the reasons set forth above, the court will grant, in

26

27

28
ORDER RE SUMMARY JUDGMENT-   217

its entirety, defendants' motion in limine as to Dr. Ivanov.[160]

With regard to the thyroid cancer case-control study, the risk estimates are not sufficiently reliable, and therefore are stricken based on Prong 1 of Daubert. If the study is offered just for the proposition that I-131 is "capable of causing" thyroid cancer, it does not "fit" and must be stricken based on Prong 2 of Daubert.

With regard to the non-cancerous thyroid disease study, the risk estimates are not reliable and therefore, are stricken based on Prong 1 of Daubert. If the study is offered just for the proposition that I-131 is "capable of causing" goiter, it is not even reliable enough for that proposition. It is also not reliable enough for the proposition that I-131 is "capable of causing" autoimmune thyroiditis.

5.   Sara Peters/Douglas Gnepp

a.   Introduction

Drs. Peters and Gnepp are pathologists. As pathologists, they study the essential nature of diseases and especially the structural and functional changes produced by them. In 1995, they co-authored a report entitled "Evaluation of Pathologic Effects of Radioiodine." The report concerns the "pathologic effects of radiation, and in particular of radioactive iodine (principally I-131) on the thyroid gland." (Peters/Gnepp Rpt. at

---

[160] The court will also deny plaintiffs' motion to strike defendants' reply on the motion in limine. Defendants' reply is entirely responsive to plaintiffs' response and does not raise any new arguments or cite any new evidence.

ORDER RE SUMMARY JUDGMENT-   **218**

p. 1).

The report discusses and details the impact of radioactive iodine on the thyroid gland. According to Peters' section of the report:

> After more than fifty years of clinical experience with therapeutic radioactive iodine, however, it is clear that a spectrum of pathologic processes **can** result from radioactive iodine exposure depending on such factors as dose, thyroid gland uptake, and individual patient characteristics including age, sex, iodine status, preexisting thyroid abnormalities and genetic makeup at the time of exposure.
>
> Animal studies and human thyroid tissue removed surgically demonstrate that the 'acute' effects of radiation exposure, i.e. those seen from approximately 6 weeks to 3 months after exposure are histologically indistinguishable from those of chronic lymphocytic thyroiditis. Histologic changes in patients with more radioactive iodine exposures include nuclear enlargement, nuclear atypia, oncocytic metaplasia, squamous metaplasia, follicular atrophy, epithelial and stromal cell atypia and fibrosis. **These changes may also be seen in a background of chronic lymphocytic thyroiditis. These findings which correlate with a clinical picture, <u>are not necessarily specific for radiation associated changes</u>.** However, vascular changes including intimal thickening and sclerosis of arterial walls . . ., often with inflammatory cell cuffing, **can be <u>relatively</u> specific changes associated with prior radiation exposure.**

(Peters/Gnepp Rpt. at p. 7)(Emphasis added).

Peters goes on to discuss a variety of health effects from radiation exposure. Among other things she states "[t]he most common clinical consequence of exposure to radioactive iodine particularly to subablative exposures[161] is hypothyroidism;"

---

[161]   Exposures that are not high enough to ablate the thyroid- i.e. vaporize or destroy it.

ORDER RE SUMMARY JUDGMENT-   219

1 animal studies show that radioactive iodine is "associated" with

2 an increased incidence of both benign and malignant thyroid

3 nodules; children treated with radioactive iodine for

4 hyperthyroidism show that both benign and malignant thyroid

5 neoplasms "may" occur in patients who receive inadequate,

6 subablative doses of radioactive iodine; pre-existing low-grade

7 thyroid carcinomas "may" undergo transformation to highly

8 aggressive anaplastic thyroid carcinomas following exposure to

9 radiation; hyperthyroidism has been "found" in a "subset" of

10 patients following radiation exposure; a number of other studies

11 have reported a variety of other abnormalities following

12 therapeutic doses of radioactive iodine including persistent

13 chromosomal damage, in situ breast carcinoma in females, germinal

14 cell dysfunction in males, and increased incidence of leukemia,

15 bladder carcinomas, salivary gland tumors and melanoma. (Id. at

16 pp. 7-8; 10).

17   Peters discusses evidence from nuclear "accidents" (A-bomb;

18 Nevada nuclear testing; and Chernobyl) which she says

19 "highlights" the "association" between radioactive iodine

20 exposure from nuclear fallout and the subsequent development of

21 both hypothyroidism and thyroid carcinomas. According to Peters:

22   I understand that the radionuclides released
            from the Chernobyl explosion and the lower
23   levels of radiation exposure from Chernobyl
            are similar to those downwind from the Hanford
24   reactor. Persons exposed to radiation from
            the Hanford reactor would be expected to have
25   radiation exposure levels similar to the lower
            levels from Chernobyl, and therefore would
26   be at similar risk for developing thyroid
            abnormalities including hypothyroidism,
27   benign thyroid nodules and goiters, and

28
ORDER RE SUMMARY JUDGMENT-   220

1
2
3

> thyroid carcinomas including clinically
> aggressive variants. A small subset of
> patients would be expected to develop hyper-
> thyroidism.

(Id. at 10-11).

4
5
6
7
8
9
10
11
12
13
14
15
16
17
18
19

Dr. Gnepp's contribution to the report concerns his work with a Russian pathologist, Dr. Yuri Nikiforov, analyzing thyroid lesions diagnosed in the population exposed to radiation from the Chernobyl accident. Gnepp discusses the increased incidence of thyroid cancer in children under age 15 residing in Belarus: 2 cases clinically diagnosed in 1986 (year of the Chernobyl accident) and 63 new cases morphologically[162] diagnosed in 1992. He says that microscopically, the tumors usually were aggressive, often demonstrating diffuse intrathyroidal tumor dissemination, thyroid capsular and adjacent soft tissue invasion, cervical lymph node metastases (88%) and rare pulmonary metastases (2%). Papillary carcinoma was diagnosed in 99% of patients with an unusually high frequency of solid growth patterns, and a rare case of follicular and medullary carcinoma was also observed. (Peters/Gnepp Rpt. at pp. 11-12).

According to Gnepp:

20
21
22
23
24
25

> In addition to high levels of radiation, in the
> region . . . of the Republic of Belarus near the
> reactor, adjacent regions were exposed to lower,
> but significant, doses of radioisotopes released
> from the explosion. Recent data have become
> available from Kaluga and Bryansk regions indicating
> there are also significant increases in the incidence
> of thyroid carcinomas, some of which are behaving
> in a biologically aggressive fashion, with frequent

26
27
28

[162] Of or relating to morphology which is the branch of biology dealing with the form and structure of animals and plants.

ORDER RE SUMMARY JUDGMENT-   221

lymph nodal (31%) and pulmonary metastases (18%).

It is my understanding that the radioisotopes released from the Hanford reactor were very similar to those released from the Chernobyl explosion, and that the lower level radiation exposures from Chernobyl are similar to those downwind of the Hanford reactor. Therefore, persons exposed to radiation from the Hanford reactor should have similar dose levels of radiation exposure to the lower level Chernobyl exposures, and therefore similar risks for developing thyroid carcinomas and other benign thyroid lesions.

(Id. at pp. 12-13).

b.   Discussion

Defendants do not dispute the qualifications of Peters and Gnepp to render an opinion about the pathogenesis[163] of thyroid cancer and thyroid disease in general. Defendants do not explicitly challenge their methodology. Rather, defendants move to exclude the Peters/Gnepp report on the basis of the fit/relevancy prong of Daubert (Prong 2). According to defendants, because the report does not contain an opinion concerning the thyroid radiation dose necessary to double the risk of any conditions claimed or the risk estimates necessary to assess causation, it does not relate to plaintiffs' causation burden of proof.

The Peters/Gnepp report clearly recognizes that radiation exposure, let alone internal radioiodine exposure, is not the only potential cause of neoplastic and non-neoplastic thyroid disease. Peters and Gnepp discuss the disease process- i.e. cellular changes- but they cannot say the process is **unique** to

---

[163]   The origination and development of a disease.

ORDER RE SUMMARY JUDGMENT-   222

1 radiation exposure.   They cannot pathologically distinguish a

2 radiation-induced cancer from a non-radiation induced cancer.

3 They cannot distinguish radiation-induced hypothyroidism from

4 non-radiation induced hypothyroidism.   Hence, Peters' comments:

5 1) these findings which correlate with a clinical picture, are

6 not necessarily specific for radiation; 2) radioactive iodine is

7 **"associated"** with an increased incidence of both benign and

8 malignant thyroid nodules; 3) children treated with radioactive

9 iodine for hyperthyroidism show that both benign and malignant

10 thyroid neoplasms **"may"** occur in patients who receive inadequate,

11 subablative doses of radioactive iodine; 4) pre-existing low-

12 grade thyroid carcinomas **"may"** undergo transformation to highly

13 aggressive anaplastic thyroid carcinomas following exposure to

14 radiation.[164]

15   Peters and Gnepp say nothing about the dose of radiation

16 necessary to double the risk of contracting any of the neoplastic

17 or non-neoplastic conditions mentioned in their report.   Indeed,

18 they offer nothing specific about the dose of radiation which is

19 "capable of causing" those conditions.[165]   They discuss dose and

20 _____

21 [164] Peters states that this "possibility" is
"controversial." It is based on her own studies and observations

22 of patients with Graves' disease who were subsequently treated
with radioactive iodine.   Peters says her studies and

23 observations "suggest" members of this group who develop thyroid
carcinomas are at risk for developing more aggressive disease

24 when compared to patients who did not receive radiation.
(Peters/Gnepp Rpt. at p. 8).

25   [165] In their response brief, plaintiffs say Dr. Peters
understands that dose uptake levels to Hanford downwinders during

26 the period 1944-70 ranged from 5-800 rads, "but that the average
dose is well over 20 rads."   This 20 rads figure is apparently

27 pulled from Ivanov's report which, according to plaintiffs, was

28 **ORDER RE SUMMARY JUDGMENT-**   **223**

1 risk in only the most vague terms: "Persons exposed to radiation

2 from the Hanford reactor would be expected to have **radiation**

3 **exposure levels** similar to the lower levels from Chernobyl and

4 therefore, would be at **similar risk** for developing thyroid

5 abnormalities . . . ."

6   There is no dispute from plaintiffs that Peters and Gnepp do

7 not provide doubling dose information. The plaintiffs, of

8 course, contend such information is irrelevant to the generic

9 causation phase of the proceedings which they say requires them

10 to prove only that I-131 is "capable of causing" neoplastic and

11 non-neoplastic diseases. According to plaintiffs, Peters and

12 Gnepp provide **relevant** information about the complex causal

13 "connections" between radioiodine and health effects and the

14 pathogenesis of benign and malignant thyroid disorders and non-

15 neoplastic thyroid disease.

16   Plaintiffs indicate Peters and Gnepp have undertaken a

17 review of thyroid pathology slides of individuals exposed to

18 Chernobyl or Hanford emissions. However, no such thing is

19 mentioned in their report. Plaintiffs say Peters can identify a

20 range of ionizing radiation induced histologic[166] features in

21 both neoplastic and non-neoplastic thyroid tissue and at the

---

reviewed by Peters. Nonetheless, the fact remains that in her
report, Peters does not specifically talk about Ivanov's report
or about a specific dose level for induction of neoplastic and
non-neoplastic diseases. Pursuant to Fed. R. Civ. P.
26(a)(2)(B), the expert's report is to contain a "complete"
statement of all opinions to be expressed and the basis and
reasons therefor, as well as the data or other information
considered by the witness in forming the opinions.

[166] Relating to tissue structure or organization.

ORDER RE SUMMARY JUDGMENT-   224

individual causation phase, she is prepared to compare these

characteristics with the clinical[167] and histologic features of

benign or malignant thyroid disorders from 141 Hanford

downwinders who were treated surgically.

It appears the sole purpose of the Peters/Gnepp report was

to discuss the pathological mechanism by which ionizing radiation

causes thyroid cancer and hypothyroidism, and is **believed** to

cause various non-neoplastic thyroid disease.[168] Plaintiffs

acknowledge as much in their brief:

> At the stage for determining individual
> causation both Peters and Gnepp will present
> evidence relating to risk estimates for
> individuals upon study of the specific
> individual risk factors involved. The jury in
> this case must ultimately determine whether the
> radiation releases from Hanford caused plaintiffs'
> injuries. **The opinions of doctors Peters and
> Gnepp are not being offered as conclusive proof
> on this ultimate issue, but are now being offered
> as a body of evidence directly relating to
> unresolved general causation issues- the causal
> relationship between radiation and thyroid
> disease, the types of thyroid illness caused by
> radiation, the etiology of autoimmune thyroiditis,
> hypothyroidism and other thyroid illnesses, the
> comparison between the Hanford experience and the
> emerging Chernobyl data and also the wealth of
> literature of other iodine/thyroid studies, and
> the other damages and illnesses caused by ingested
> iodine 131.**

(Plaintiffs' Response Br. at p. 7)(Emphasis added).

Evidence of the pathological mechanism by which radiation

---

[167] Direct observation of the patient.

[168] Defendants agree the scientific consensus is that
ionizing radiation is capable of causing thyroid cancer and non-
autoimmune hypothyroidism (at least at high doses). The question
has not been as clearly resolved for non-neoplastic conditions
other than non-autoimmune hypothyroidism- i.e. thyroiditis,
hyperthyroidism, etc.

ORDER RE SUMMARY JUDGMENT-   225

1   causes or is believed to cause certain diseases is clearly

2   relevant to the issue of the capability of radiation to cause

3   those diseases. The problem is that such evidence, by itself, is

4   insufficient to sustain the plaintiffs' ultimate burden of proof-

5   whether radiation exposure is a "more likely than not" cause of

6   their diseases.

7   Plaintiffs cite Ambrosini v. Labarraque, 101 F.3d 129 (D.C.

8   Cir. 1996). In that case, the court found testimony by an

9   epidemiologist that Depo-Provera "could cause" birth defects did

10 not warrant exclusion under Daubert simply because it failed to

11 establish the causal link to a specified degree of probability.

12 According to the D.C. Circuit, that the testimony of the

13 epidemiologist might be insufficient for the plaintiffs to

14 survive summary judgment did not "necessarily" defeat its

15 admissibility under Daubert's fitness prong. Because his

16 testimony was sufficiently tied to the facts at issue, the D.C.

17 Circuit concluded the fitness prong was satisfied. Id. at 135-

18 36.

19   Nonetheless, in Ambrosini the court also found admissible

20 the testimony of a teratologist that to a "reasonable medical

21 certainty" Depo-Provera had caused the birth defects of plaintiff

22 Teresa Ambrosini. That evidence on "specific causation," in

23 combination with the epidemiologist's testimony on general

24 causation, provided "sufficient" evidence to raise an issue of

25 material fact as to the cause of Teresa Ambrosini's birth

26 defects. Id. at 141.

27   Testimony that iodine-131 is "capable of causing" neoplastic

28 **ORDER RE SUMMARY JUDGMENT-   226**

1 and non-neoplastic thyroid disease is not sufficient for the

2 plaintiffs to survive summary judgment and get their cases to

3 trial. They need additional evidence which at least raises an

4 inference that the causal link is established to a specified

5 degree of probability. In this case, that requires a showing it

6 is "more likely than not" that radiation exposure caused the

7 particular diseases in question. Without that component,

8 plaintiffs cannot now or ever meet their burden of proof. There

9 is no reason to let a jury hear evidence that radiation is

10 "capable of causing" a disease unless they are also going to hear

11 admissible evidence that it is "more likely than not" a cause of

12 the disease. Without the total package, evidence that radiation

13 is "capable of causing" a disease is inadmissible.

14   In Daubert II, the Ninth Circuit observed the distinction

15 between general relevancy under FRE 402 and the fit/relevancy

16 requirement of FRE 702:

17   The Supreme Court [in Daubert I] recognized
           that the "fit"' requirement "goes primarily to
18   relevance," but it obviously did not intend
           the second prong of Rule 702 to merely be
19   a reiteration of the general relevancy
           requirement of Rule 402. In elucidating the
20   "fit" requirement, the Supreme Court noted
           that scientific expert testimony carries
21   special dangers to the fact-finding process
           because it "'can be both powerful and quite
22   misleading because of the difficulty in
           evaluating it. . . .'" Federal judges must
23   therefore exclude proffered scientific
           evidence under Rules 702 and 403 unless they
24   are convinced that it speaks clearly and
           directly to an issue in dispute in the case,
25   and that it will not mislead the jury.

26 Daubert II, 43 F.3d at 1321, n. 17.

27   Unless radiation is "capable of causing" a disease, it

28 **ORDER RE SUMMARY JUDGMENT-   227**

1 cannot be a "more likely than not" cause of that disease. Thus,

2 evidence that radiation is "capable of causing" a certain disease

3 is **generally** relevant to the causation analysis as a whole.

4 However, by itself, it is insufficient to sustain a jury verdict.

5 Presented by itself to a jury, such complex scientific evidence

6 could mislead a jury into thinking it could render a plaintiffs'

7 verdict based on such evidence alone. That is definitely not the

8 case. Such evidence does not speak clearly and directly to the

9 ultimate issue in dispute- whether it is "more likely than not"

10 that radiation exposure is a cause of the disease.

11   Drs. Peters and Gnepp cannot tell a jury to a reasonable

12 pathological certainty that radiation exposure caused diseases in

13 particular individuals. All they can do is speak in general

14 about the pathological mechanism by which radiation causes or is

15 believed to cause the disease.[169] Other evidence is needed to

16 establish an inference it is "more likely than not" radiation is

17 a cause. Pathological evidence is insufficient to supply that

18 inference. Only epidemiological evidence can supply that

19 inference through scientifically reliable risk estimates.

20   Peters has submitted a post-report affidavit (Foulds Ex.

21 89). In that affidavit, she indicates that for the past two

22 years she has reviewed histologic sections of thyroid

23 (microscopic slides), patient medical records and other studies

24 _____

25   [169] Accordingly, considering only the **contents of their**
   report, there is nothing additional they will be able to offer at
26 the individual causation phase to show that a particular
   plaintiff's cancer or thyroid disease was induced by I-131 as
27 opposed to something else. However, see n. 175 _infra_.

28 **ORDER RE SUMMARY JUDGMENT-   228**

1 from 141 Hanford downwinders who have been diagnosed with benign

2 and/or malignant thyroid disorders. In addition to confirming

3 the original diagnoses, she says she has noted several

4 similarities to the clinical and histopathologic materials from

5 Chernobyl, chief among which is age at exposure. According to

6 Peters, the vast majority of Hanford downwinders who subsequently

7 developed thyroid carcinomas were exposed to I-131 in utero, in

8 childhood, adolescence, or early adulthood. (Id. at Paragraph

9 L). Peters adds:

10   From their clinical histories and other
            medical records, I have been able to identify
11   the geographic location of those downwinders
            whose records I have examined. From other
12   documents concerning Hanford I-131 dose
            reconstruction, I understand that these
13   downwinders are estimated to have been exposed
            to between 10 and 200 rads.
14
            Persons exposed to radiation from the Hanford facility
15   are, therefore, estimated to have radiation
            exposure levels similar to lower levels from
16   Chernobyl and would, therefore, be at similar
            risk of developing similar types of thyroid
17   abnormalities including hypothyroidism, benign
            thyroid nodules, goiters and carcinomas, including
18   clinically aggressive variants. The most common
            result of low dose exposure is reportedly hypo-
19   thyroidism. I am advised that to date there are
            555 cases of hypothyroidism in the Evenson client
20   base, 209 cases of hyperthyroidism, 162 cases of
            thyroid carcinoma and 241 cases of other thyroid
21   disorders.

22 (Id. at Paragraphs M and N).

23   Peters still does not say anything specific about risk,

24 although she refers to a dose level between 10 and 200 rads. She

25 does not explain exactly where she obtained this dose range.

26 There is **no** mention of a specific dose level in Peters' **report**.

27 Peters' affidavit appears an attempt to make up for the

28 **ORDER RE SUMMARY JUDGMENT-   229**

conclusory assertion in the report that persons exposed to

Hanford emissions would be expected to have **radiation exposure**

levels similar to the lower levels from Chernobyl and therefore,

be at **similar risk** for developing thyroid abnormalities.[170] The

report says nothing about the comparison of Chernobyl data with

histologic sections of thyroid (microscopic slides), patient

medical records and other studies of Hanford downwinders.[171]

For this reason, defendants contend Peters' affidavit should be

stricken as an improper or untimely effort to cure the

deficiencies of her original report.

It is not necessary to strike Peters' affidavit. One reason

is the affidavit still does not establish the relationship

between radiation dose and risk necessary to present claims to a

jury. The affidavit essentially says no more than what the

report says: these are the pathological mechanisms by which

radiation causes or is believed to cause certain types of

damages. As pathologists, that is all Peters and Gnepp are

qualified to discuss. In any event, Fed. R. Civ. P. 26(a)(2)(B)

limits Peters and Gnepp to the contents of their **report** and the

---

[170] Gnepp's section of the report is also deficient in this
respect. As defendants point out, he specifies no basis for
comparing Chernobyl and Hanford emissions in terms of health
effects.

[171] Nor does the report or the affidavit explain how
comparison of Chernobyl and Hanford is going to help determine
whether Hanford emissions are a "more likely than not" cause of
particular diseases. Here again, plaintiffs may argue the
comparison is only intended to prove that Hanford downwinders
were exposed to doses known to be "capable of causing" certain
conditions. However, the report does not even accomplish that
because of the conclusory comparative analysis contained therein.

ORDER RE SUMMARY JUDGMENT-   230

1 opinions expressed therein.

2   In the final analysis, the question is how does the

3 Peters/Gnepp **report** benefit the plaintiffs, if at all.

4 It is relevant and admissible evidence for the very general

5 proposition that radiation is "capable of causing" thyroid cancer

6 and hypothyroidism (at least the direct cell-killing version).

7 Presumably, defendants do not take issue with this basic

8 proposition, although the requisite dose level is another

9 issue.[172]

10   The report does not provide scientifically reliable (and

11 admissible) evidence for the proposition that radiation exposure

12 is "capable of causing" the transformation of pre-existing low

13 grade thyroid carcinomas into highly aggressive anaplastic[173]

14 thyroid carcinomas. As noted above, all Peters offers in support

15 of this are her own observations and studies which "suggest" that

16 individuals with Graves' disease who are subsequently treated

17 with radioactive iodine develop more aggressive disease when

18 compared to patients who did not receive radiation.

19   The report does not offer scientifically reliable (and

20 admissible) evidence for the proposition that radiation is

21 _____

        [172] Defendants assert the scientific literature at best
22 establishes a high dose causal connection between radiation and
    non-autoimmune hypothyroidism. Apparently, defendants are
23 unwilling to accept the existence of a non-threshold dose level
    for thyroid cancer (that exposure can cause cancer down to the
24 very lowest doses). Defendants accurately note that the
    Peters/Gnepp **report** says nothing about a non-threshold dose level
25 for thyroid cancer. Peters' affidavit (Paragraph P) discusses
    the notion, but the report is what counts.
26
        [173] Reversion of cells to a more primitive or
27 undifferentiated form.
28
    ORDER RE SUMMARY JUDGMENT-   231

"capable of causing" hyperthyroidism or that radioactive iodine
is "capable of causing" chromosomal damage, in situ breast
carcinoma in females, germinal cell dysfunction in males,
increased incidence of leukemia, bladder carcinomas, salivary
gland tumors and melanomas. The report offers only the barest
conclusions that hyperthyroidism is "found" in a "subset" of
patients following radiation exposure and that a "number of other
studies have reported a variety of other abnormalities following
therapeutic doses of radioactive iodine," including chromosomal
damage, in situ breast carcinoma in females, germinal cell
dysfunction in males, increased incidence of leukemia, bladder
carcinomas, salivary gland tumors and melanomas.[174] At least
with regard to thyroid cancer and hypothyroidism, the report
discusses the specific pathological mechanisms associated with
those diseases. Not so with hyperthyroidism and these "other
abnormalities."

With regard to autoimmune thyroiditis, the report indicates
that follow-up data from Chernobyl have documented an increase in
antimicrosomal antibodies in the sera of exposed children. The
report states these antibodies correlate with histologic findings

_____

[174] Even if it were permissible to go beyond the report,
plaintiffs' brief and Dr. Peters' affidavit do not remedy the
deficiency here. Plaintiffs' brief asserts that one of the
defense experts in this action, Dr. Lars Erik Holm, discusses the
"possibilities" that pre-existing low grade carcinomas may
undergo transformation to highly aggressive anaplastic thyroid
carcinomas following radiation exposure. In her affidavit,
Peters refers to a recent study by Dr. Holm finding an
"association" between radiation exposure and lower GI
(gastrointestinal) cancers. (Peters Affidavit at Paragraph H).
An "association" is not necessarily causal. "Reference Guide on
Epidemiology" at p. 147.

ORDER RE SUMMARY JUDGMENT-   232

1   of chronic lymphocytic thyroiditis and in the appropriate

2   clinical setting are diagnostic of autoimmune thyroiditis.[175]

3   The report adds that the end stage of any chronic lymphocytic

4   thyroiditis is characterized histologically by follicular atrophy

5   and fibrosis and clinically by hypothyroidism. However, nowhere

6   in the **report** does Peters opine that radiation causes antibodies

7   or autoimmune thyroiditis. Therefore, the **report** does not

8   constitute scientifically reliable (and admissible) evidence that

9   radiation is "capable of causing" autoimmune thyroiditis.

10

11   **c. Conclusion**

12   The court will grant defendants' motion in limine, exclude

13   the Peters/Gnepp report, and exclude them from testifying at

14   trial **about matters in the report**. Their testimony does not

15   "fit" (i.e. is not relevant to) the burden of proof which

16   plaintiffs must ultimately carry.[176] Furthermore, for the

17   _____

18   [175]   Plaintiffs cite this same evidence as support for Dr.
Ivanov's proposition that radiation is "capable of causing"
autoimmune thyroiditis at doses as low as 20 rads. However, as

19   noted, the increase of antimicrosomal antibodies, although
indicative of autoimmune thyroiditis, is not necessarily

20   synonymous with a diagnosis of autoimmune thyroiditis.
Accordingly, the evidence of increase of antibodies in the seras

21   of children exposed to Chernobyl emissions is not enough to
overcome Ivanov's finding that the incidence of **diagnosed**

22   autoimmune thyroiditis was higher in his control group than in
his case group.

23

24   [176]   A question arises as to whether Peters and/or Gnepp
should be able to testify on behalf of individual plaintiffs who
meet the doubling doses for thyroid cancer (including nodules and

25   adenomas) and non-autoimmune hypothyroidism (clinical and
subclinical). For those individuals, an inference is raised that

26   their thyroid cancer or hypothyroidism is "more likely than not"
the result of their exposure to Hanford iodine emissions. Among

27   those individuals may be some of the 141 downwinders whose tissue

28   **ORDER RE SUMMARY JUDGMENT-   233**

proposition that radiation is "capable of causing" certain

conditions (hyperthyroidism, non-thyroid cancers, leukemia,

chromosomal damages, germinal cell dysfunction in males, and

autoimmune thyroiditis), the Peters/Gnepp report is not

scientifically reliable. Those opinions warrant exclusion on the

basis of Prong 1 of <u>Daubert</u>.

The court will deny plaintiffs' motion to strike defendants'

reply regarding the Peters/Gnepp motion in limine. The reply is

wholly responsive and does not unfairly raise any new arguments.

It reiterates arguments contained in defendants' opening brief

and responds to specific points raised in plaintiffs' response

brief.

6.   Richard Clapp/R-11 Survey

a.   Introduction

The R-11 Survey was a telephone survey conducted between

1992 and 1995, the purpose of which was to obtain information

about the prevalence of thyroid diseases among graduates of

fourteen high schools in areas downwind of the Hanford facility.

The survey attempted to include all graduates between 1950 and

---

Dr. Peters has actually examined. Presumably, Peters would
testify about the pathological mechanisms she has observed in the
tissue and that it is consistent with radiation exposure.
     Defendants may argue such testimony is still irrelevant
because it does not make it any "more likely than not" that
radiation exposure is the culprit. On the other hand, testimony
about the particular pathological mechanism at work, along with
other testimony (medical and non-medical) ruling out other
potential causes, may be relevant to a jury's determination of
whether causation in fact is established.

ORDER RE SUMMARY JUDGMENT-   234

1969. (Clapp 1995 Rpt. at p. 5). The results from the survey
were compared to the results of the 1993 National Health
Interview Survey (NHIS). The comparison yielded the following
results (for goiter and other non-neoplastic thyroid disorders):

Age Category	R-11 Survey Rate	NHIS Rate
18 to 44	144.5/1000[177]	14.1/1000
45 to 64	181.4/1000	26.7/1000

Dr. Richard Clapp, an epidemiologist with the JSI Center for
Environmental Health Studies, analyzed the data of the R-11
Survey. In his 1995 report, Clapp reached the following
conclusion:

> . . . there is considerably more goiter and
> other diseases of the thyroid reported in the
> survey respondents than in national survey data.
> . . . These preliminary calculations indicate
> that R-11 Survey respondents report goiter and other
> diseases of the thyroid approximately six to
> ten times as frequently as respondents in the
> latest NHIS survey.

(Clapp 1995 Rpt. at p. 6).

In April 1996, Clapp prepared a "Supplemental Report of R-11
Survey Results." In this report, he offered an opinion about the
prevalence of thyroid cancer. Based on 22 cases of thyroid
cancer out of a total of 7,366 cases, a "crude" thyroid cancer
prevalence estimate of 298.7 per 100,000 was derived. In order
to "refine" the prevalence estimate, age-specific prevalence
rates were taken from the Connecticut Tumor Registry Data.

---

[177] Rate of thyroid disease per 1000 cases.

ORDER RE SUMMARY JUDGMENT-   235

According to Clapp:

> Using the published prevalence rates for
> males and females in age groups 30-49 and
> 50-59, combined prevalence rates for the
> total population of males and females were
> estimated by averaging the sex-specific
> rates. These prevalence rates from the
> Connecticut data were then multiplied by
> the number of respondents in the corresponding
> age group in the survey to get the expected
> prevalence in the R-11 population.

(Clapp 1996 Rpt. at p. 2).

Clapp's calculations produced the following expected number

of thyroid cancer cases for the two age groups and the total

expected in the (R-11) survey population as a whole:

Age Group	Prevalence Rate	R-11 Population	Expected No.
30-49	72.6/100,000	4582	3.3
50-59	99.8/100,000	2784	2.8
			TOTAL= 6.1

Dividing the 22 "observed" number of thyroid cancer cases,

"based on self-report and record review," by the 6.1 expected

cases resulted in a prevalence rate ratio estimate of 3.6. In

other words, the result was a three-fold excess of thyroid cancer

among the R-11 survey respondents as compared to the Connecticut

Tumor Registry Data. (Clapp 1996 Rpt. at p. 3).

Clapp also came up with some prevalence estimates for breast

cancer, lung cancer and leukemia. However, because there were

"limited medical records available for review of these reported

cases, . . . it was not possible to verify the diagnosis of the

ORDER RE SUMMARY JUDGMENT-   236

1 majority of the reported cases of these three types of cancer."

2 Nevertheless, added Clapp, if the reporting was as accurate as

3 for thyroid cancer, the data represented "a nearly three-fold

4 excess prevalence of breast cancer, a greater than four-fold

5 excess of lung cancer, and a greater than ten-fold excess of

6 leukemia compared to published prevalence in Connecticut."

7 (Id.)[178]

8   Clapp's conclusion was as follows:

9   This excess of diseases and cancer of the thyroid
          is highly significant. The magnitude and timing[179]

10   of the excess is consistent with the results of
          other published studies of exposed populations

11   in other parts of the U.S and elsewhere. **Assuming
          that this population responding to the R-11**

12   **survey was exposed to substantial amounts of ionizing
          radiation from iodine and other radionuclides, it**

13   _____

14   [178] In a September 5, 1997 letter (Foulds Ex. 144), Dr.
     Richard Bird informed plaintiffs' counsel that for breast cancer,

15   45 records had been reviewed at the time of the April 1996 report
     with 44 cases confirmed from review of the medical records; for

16   lung cancer, 11 records had been reviewed with 11 cases
     confirmed; for leukemia, four records had been reviewed with four

17   cases confirmed. According to Bird:

18   It can be expected, based on this finding
          of very high confirmation of those cancer

19   cases reviewed, that the remainder of the
          cases from the survey, of those not yet

20   reviewed, will also have a very high
          confirmation rate.

21
     This is speculation on Dr. Bird's part and does not change

22   the concern expressed by Dr. Clapp in his April 1996 report that
     it is still not possible, **at this time**, "to verify the diagnosis

23   of the **majority** of the reported cases of these three types of
     cancer."

24
       [179] According to Clapp, the number of reported thyroid

25   disease cases increased steadily from the mid-1940s to the mid-
     1960s, and then declined somewhat throughout the 1970s and 1980s.

26   (Clapp 1996 Rpt. at pp. 3 and 4). The majority of radioiodine
     was emitted from Hanford during the period between the mid-40s

27   and the mid-60s.

28
     ORDER RE SUMMARY JUDGMENT-   237

1   **is my opinion that the observed excess thyroid**
         disease and thyroid cancer, to a reasonable degree
2   **of scientific certainty, was caused or contributed to**
         by the exposure.
3
(<u>Id</u>. at p. 4)(Emphasis added).
4

5   **b.   Fit/Relevancy**

6   Defendants assert the R-11 Survey is irrelevant to this case

7   because it does not address the "core" issues of dose and

8   causation. According to defendants, even accepting Clapp's

9   opinion at face value, the R-11 Survey does not provide any

10 evidence of a causal connection between Hanford emissions and

11 plaintiffs' claims, and does not provide any basis for analyzing

12 causation. The court agrees.

13   Plaintiffs' "generic causation" burden is to produce

14 evidence showing at what radiation dose the risk of contracting a

15 disease is doubled (i.e. a "more likely than not" cause of the

16 disease). Clapp's opinion is irrelevant because it says nothing

17 about dose or risk. Without dose information, there is no way to

18 tell whether an increased prevalence of disease among Hanford

19 downwinders is "more likely than not" due to radiation as opposed

20 to any number of other potential sources. Clapp concedes as much

21 by "assuming" that **if** the R-11 Survey respondents were exposed to

22 "substantial" amounts of ionizing radiation, any excess thyroid

23 disease and thyroid cancer was caused or contributed to by such

24 exposure.

25   Plaintiffs admit Clapp and the R-11 Survey provide no

26

27
28
    ORDER RE SUMMARY JUDGMENT-   238

1 radiation dose information.[180] Nonetheless, plaintiffs contend

2 Clapp's testimony and the R-11 Survey is "highly relevant" as it

3 "provides direct tangible evidence that **whatever the dose may**

4 **have been**, it was enough to increase the incidence of some

5 radiogenic illnesses in the area where the plaintiffs lived." Of

6 course, what plaintiffs mean is that this evidence is relevant to

7 what they perceive to be their "generic causation" burden of

8 proof: is radiation "capable of causing" the diseases in

9 question.[181]

10   The plaintiffs say the R-11 Survey "provides evidence that

11 exposure to radiation causes thyroid problems." They add that

12 the R-11 Study is not being offered as "conclusive proof alone"

13 on the "ultimate issue" of whether plaintiffs' exposure to

14 radiation released by defendants caused their [plaintiffs']

15 injuries. Instead, plaintiffs state the R-11 Survey is "being

16 offered along with other relevant evidence that is relevant to a

17 number of the sub-issues that the jury must resolve." Among the

18 other relevant evidence is **"dose reconstruction evidence."**

19 According to plaintiffs, this evidence will allow a jury to

20 reasonably infer the source of radiation exposure for R-11 Survey

21 respondents and the plaintiffs, who come from many of the same

22

---

23   [180] "The purpose of the R-11 Study was not to recreate dose,
nor was it possible to attempt a dose response analysis."
24 (Plaintiffs' Response Br. at p. 36).

25   [181] According to plaintiffs, "Dr. Clapp does not have to
provide any opinion as to causative risk estimates (doubling
26 dose) to prove generic causation sufficient to overcome summary
judgment at this stage in the litigation." (Plaintiffs' Response
27 Br. at p. 39).

28

ORDER RE SUMMARY JUDGMENT-   239

communities, was the Hanford facility.

All of this adds up to an admission by plaintiffs that the R-11 Survey, **by itself**, cannot prove Hanford radiation emissions were a "more likely than not" cause of any individual's disease. A greater prevalence of disease in an assumedly exposed population versus an unexposed population does not necessarily mean the excess is attributable to radiation exposure as opposed to other factors.[182] Plaintiffs assert that unless defendants can convincingly explain how "other factors may have been responsible for the extraordinary incidence of thyroid problems among the R-11 Study Group," a jury can reasonably infer that "the increase in thyroid problems among that group was caused by exposure to some form of radiation." Nonetheless, plaintiffs acknowledge the existence of other potential factors, as well as the fact that ionizing radiation from Hanford emissions is not the only potential source of radiation exposure.[183]

Plaintiffs argue that "[i]f an individual living in a community downwind of a nuclear facility that has admittedly released large amounts of radiation is suffering from thyroid

_____

[182] This is similar to Dr. Ruttenber's citation to "doubling of disease rates" which Ruttenber acknowledged is not the same as a doubling of **background** incidence. It is from the background incidence of the disease that risk estimates are derived.

[183] Plaintiffs acknowledge the R-11 Study does not provide the information necessary to determine the risk that an individual's disease was due to Hanford radiation as opposed to some other source. At p. 37 of their response brief, plaintiffs state that from the R-11 Study, "a jury could reasonably conclude not only that plaintiffs were exposed to excess amounts of radiation, but that this exposure was the cause of **some** of their injuries." (Emphasis added).

ORDER RE SUMMARY JUDGMENT-   240

1 problems, [the R-11 Study] makes it **far more likely** than it would

2 be without such evidence that the radionuclides released from

3 Hanford were responsible for that individual's illness."

4 (Plaintiffs' Response Br. at p. 13)(Emphasis added). However,

5 plaintiffs cannot say it makes it **"more likely than not."**

6   A jury entrusted with the responsibility of determining

7 whether an individual's disease was "more likely than not" caused

8 by Hanford radiation emissions will not be assisted by the R-11

9 Survey which provides no information about dose and risk. That

10 information is critical to an assessment of causation. Indeed,

11 the R-11 Survey could easily mislead a jury into thinking

12 causation is established merely by prevalence of disease in the

13 Hanford environs.

14   Defendants' motion in limine will be granted on the basis of

15 Prong 2 of <u>Daubert</u>. Clapp's opinion and the R-11 Survey do not

16 "fit" and are not relevant to plaintiffs' burden of proof.[184]

17 _____

[184] The R-11 Survey is very similar to the mortality study
18 which was proffered in the TMI litigation. Dr. Steven Wing, the
    author of the study, acknowledged he was not offering an analysis
19 of the association between dose and mortality. Consequently, he
    could not make any direct correlation between the TMI accident
20 and certain increased mortality trends. The court struck his
    testimony on the basis of "fit" because it would not help the
21 trier of fact understand any fact in issue. <u>In re TMI Litigation
    Cases Consolidated II</u>, 911 F. Supp. 775, 819-20 (M.D. Pa. 1996).
22   Interestingly, the R-11 "Design Protocol" mentions the
    possibility of Dr. Wing performing the epidemiological analysis
23 of the R-11 data either by himself or with Dr. Clapp. (Clapp
    1995 Rpt. at p. 8 under heading "Verification of Radiogenic
24 Illnesses"). There is no indication, however, that Wing
    actually participated in the epidemiological analysis.
25   Clapp cannot make any "direct correlation" between Hanford
    radiation emissions and the increased prevalence of thyroid
26 disease. His opinion is contingent on an assumption the R-11
    population was exposed to "substantial" amounts of ionizing
27 radiation from iodine and other radionuclides.

28
   ORDER RE SUMMARY JUDGMENT–   241

c. Reliability

Defendants contend the R-11 Survey suffers from a myriad of methodological flaws, which in combination, render its results unreliable. They begin by pointing out the R-11 Survey is an "ecological" study. Dr. Clapp acknowledges this is so. (Clapp Affidavit, Foulds Ex. 20, at p. 4).

Studies that collect data about the group as a whole are called "ecological" studies. Such studies are useful for identifying associations, but are generally regarded as "weak" by epidemiologists. "Reference Guide on Epidemiology" at pp. 132-33. The Reference Guide offers an example of an "ecological" study which shows the limitations of such a study (some of which are discussed above in the "fit/relevancy" section):

> If [a] researcher is interested in determining
> whether a high dietary fat intake is associated
> with breast cancer, he or she can compare different
> countries on the basis of their average fat intakes
> and their average rates of breast cancer. If a
> country with a high average fat intake also tends
> to have a high rate of breast cancer, the findings
> would suggest an association between dietary fat
> and breast cancer. **However, such a finding would
> be far from conclusive because it lacks particularized
> information about an individual's exposure and disease
> status (i.e. whether an individual with high fat
> intake is more likely to have breast cancer). In
> addition to the lack of information about an
> individual's intake of fat, the researcher does
> not know about alternative individual exposures
> to other agents (or family history) that may also
> be responsible for the increased risk of breast cancer.**
> The lack of particularized information about an
> individual's exposure to an agent and disease status
> detracts from the usefulness of the study and can
> lead to an erroneous inference about the relation-
> ship between fat intake and breast cancer, known as
> an ecological fallacy. However, the study is useful

ORDER RE SUMMARY JUDGMENT-   242

1
2

> in that it identifies an area for further research:
> the fat intake of individuals who have breast cancer
> as compared with the fat intake of those who do not.

3   ("Reference Guide on Epidemiology" at p. 133)(Emphasis added).

4   The "Reference Guide" is in accord with the view of the

5   National Academy of Science Committee on Radiation Dose

6   Reconstruction for Epidemiological Uses which states that

7   "ecological studies are usually regarded as hypothesis generating

8   at best, and their results must be regarded as questionable until

9   confirmed with cohort or case-control studies."   National

10   Research Council, **Radiation Dose Reconstruction for Epidemiologic**

11   **Uses**, at p. 70 (1995).[185]

12   Clapp's response is that ecological studies are not

13   necessarily "weak," particularly when dealing with area-wide

14   exposure.   He asserts that defendants' criticisms and citation to

15   the National Research Council publication are inappropriate

16   because the purpose of the R-11 Survey was not to "generate

17   quantitative estimates of risk . . . ."[186]   (Clapp Affidavit at

18   pp. 4-5).

19   The fact the R-11 Survey is an "ecological" study does not

20   make it per se unreliable, although it may affect the "weight" it

21   should be given by a trier of fact.   The methodological soundness

22   of the survey and its reliability must be evaluated based on the

23   **purpose** for which its results are offered.   As noted, plaintiffs

24   _____

25   [185]   Defendants' Ex. 92.

26   [186]   This is further confirmation that the R-11 Survey alone
is not sufficient for plaintiffs to meet the "more likely than
27   not" evidentiary standard.

28
ORDER RE SUMMARY JUDGMENT-   243

1 concede the R-11 Survey is not offered to prove quantitative

2 estimates of risk. Rather, Clapp **hypothesizes**[187] that

3 "assuming" the R-11 survey population was exposed to

4 "substantial" amounts of ionizing radiation from iodine and other

5 radionuclides, the observed excess thyroid disease and thyroid

6 cancer, "to a reasonable degree of scientific certainty," was

7 caused or contributed to by such exposure. Whether the

8 hypothesis is borne out depends in part on the "dose

9 reconstruction evidence."[188] Even then, plaintiffs concede the

10 R-11 Survey cannot prove it is "more likely than not" that any

11 individual's thyroid disorder was caused by exposure to Hanford

12 emissions. According to plaintiffs, the R-11 Survey is only part

13 of the overall evidence supporting causation.

14 //

15

_____

16   [187] A hypothesis is an assumption or concession made for the
sake of argument. It implies insufficiency of presently
17 attainable evidence and therefore, a tentative explanation.

18   [188] In his April 1996 report, Clapp claims the prevalence
rate ratio estimate of 3.6 for thyroid cancer among the R-11
19 study subjects is similar to the relative risk estimate of 3.4
for thyroid neoplasms in individuals who were downwind from the
20 Nevada Test Site between 1951 and 1958 **and were exposed to doses
greater than 400mGy."** (Clapp 1996 Rpt. at p. 3, citing a cohort
21 study performed by Kerber, et al., 1993). Clapp later asserts in
an October 1997 letter to plaintiffs' counsel (Ex. E to Foulds
22 Reply re Motion to Strike) that the results of the Kerber cohort
study confirm "the risk estimate for this disease in the R-11
23 Survey."
        In his April 1996 report, **Clapp did not provide a "risk
24 estimate."** He acknowledged that was not the purpose of the R-11
"ecological" survey. In the Kerber study, a risk estimate was
25 provided because there obviously was specific dose information
(400 mGy). This is not the case with the R-11 Survey. Clapp's
26 opinion regarding causation depends on the assumption there was
exposure to "substantial amounts of ionizing radiation from
27 iodine and other radionuclides."
28
ORDER RE SUMMARY JUDGMENT-   244

1   **(1) Survey Design and Protocol**

2   The defendants assert the R-11 Survey was poorly designed.

3   One of the reasons say defendants is that Clapp had very little

4   involvement with the survey until he endorsed it in his November

5   1995 report.

6   At his deposition, Clapp testified he spent four hours on

7   the project in 1991, three hours in 1992, and 11 hours on

8   November 13, 1995 preparing his November 14, 1995 report. (Clapp

9   Dep. at p. 89). According to him, the R-11 Survey was already

10   designed and implementation of it had already started before he

11   began work on the project. Asked if he had any role in the

12   design of the survey, Clapp said that in late 1991 or early 1992

13   he recommended that "a national comparison be used as an

14   alternative to a comparison between exposed and non-exposed

15   groups in the Washington, Oregon, Idaho area." (Id. at pp. 34-

16   35).[189] Clapp testified that of the individuals he knew, he

17   thought Pam Metcalf was the most knowledgeable person concerning

18   the design of the R-11 Survey, although there was a Doctor

19   Cummins who **might** be "even more knowledgeable." (Id. at pp. 37-

20   38). According to Clapp, he had several conversations with

21   Metcalf in which he "stressed" that interview questions "should

22   be asked the same way every time." They also discussed "medical

23   verification of diagnoses." (Id. at p. 37).

24   Clapp was not at all involved in the interview process; he

25   did not review certain correspondence sent to study subjects

26   _____

     [189] Clapp did not say specifically the NHIS Survey. He

27   referred generically to a "national comparison."

28   **ORDER RE SUMMARY JUDGMENT-   245**

1   introducing the study and explaining it to them (Id. at 107); and

2   he did not take any steps to insure study subjects would not have

3   contact with anyone involved in the litigation (Id. at 103).

4   Clapp said he had a discussion with Metcalf that the interview

5   should not reference the ongoing litigation. According to Clapp,

6   this discussion took place before the "vast bulk" of the

7   interviews were completed. (Id. at 103-04).

8   Ms. Metcalf has no education, training or experience in

9   epidemiology. She has not submitted an expert report and is not

10 an expert in this case. According to defendants, despite

11 warnings the survey should be separate from the litigation and

12 that "no one remotely connected with the associated counsel

13 should be involved with the process of data analysis nor even

14 connected with the raw data itself," (Alexandra R. Fleetwood,

15 Proposal R-11 Survey Project at p. 2)[190], plaintiffs' counsel

16 was involved with the design of the survey and in the decision to

17 drop the original control group after more than a thousand

18 interviews had been conducted with R-11 control group subjects.

19   At his deposition, Clapp stated his belief that three people

20 were involved in the design of the survey- Metcalf, Cummins, and

21 Tom Foulds. However, he could not specify Foulds' role. (Clapp

22 Dep. at p. 39). Foulds chimed in that his involvement was

23 financial. (Id.) Clapp testified the decision to drop the

24 control towns was his "in consultation with Miss Metcalf and with

25 Mr. Foulds." (Id. at p. 140).

26 _____

27   [190] Defendants' Ex. 32.

28 **ORDER RE SUMMARY JUDGMENT-   246**

1   Plaintiffs assert Metcalf was not one of the "designers" of

2 the study and any work done by her "was primarily to computer

3 format the input she received from Dr. Cummins." In an

4 affidavit, Metcalf describes the genesis of the R-11 Survey:

5   The Hanford Downwinder Coalition had a variety
              of health effects type questions that they wanted
6   to include in a survey so as to better understand
              health patterns which may be related to their
7   exposure. These questions were approved by Dr.
              Wally Cummins, Ph.D., Research Director with
8   Profiles NW, and the principal investigator for
              the Hanford Health Effects Study of Military
9   Personnel, who was concurrently conducting a Hanford
              Veterans Study. I next met with Dr. Cummins as
10   well as Mr. Foulds, whose group was to provide
              funding for the R-11 study. I did not participate
11   in the design of any study questions other than
              to give additional input to the Hanford Downwinders[']
12   health needs and to format the final material into
              the database survey questionnaire. The overall
13   design protocol was in place before any actual
              survey work began.

14

(Metcalf Affidavit at p. 2, Foulds Ex. 76).

15
    Metcalf's affidavit suggests Dr. Cummins was the primary
16
force behind the design of the R-11 Survey. This Dr. Cummins is
17
a mystery man. He is not an expert in this case; has not
18
submitted an expert report, or even an affidavit regarding his
19
alleged role in the design of the R-11 Survey. There is no
20
evidence by which the court can judge his qualifications.[191] An
21
affidavit from Judith Jurji, former president of the Hanford
22
Downwinders Coalition, says she understood "Dr. Wally Cummings,
23
PhD, . . . **would** help collaborate with our efforts, both in
24
formalizing our health questionnaire and carrying out other
25

26
_____

    [191] In their response brief, plaintiffs assert Cummins is a
27 "qualified epidemiologist." (Response Br. at p. 23).
28
ORDER RE SUMMARY JUDGMENT-   247

1 concerns pertaining to health manifestations and patterns, into

2 the R-11 survey questionnaire."   (Jurji Affidavit at p. 3, Foulds

3 Ex. 67)(Emphasis added).

4   The affidavits of Metcalf and Jurji do not state that

5 Cummins (or "Cummings," as the case may be) drafted the

6 questions, when he would have approved them, or that he even

7 approved the study design.   To top it all off, Clapp never spoke

8 with Cummins (Clapp Dep. at p. 36); Cummins is not mentioned in

9 Clapp's report or affidavit; and the only knowledge Clapp had of

10 Cummins was provided to him by plaintiffs' counsel (Foulds) who

11 told him Cummins had been involved in the design stage. (Id. at

12 96).   Excluding Cummins, there was no one with an epidemiological

13 background involved in the design of the survey.   Metcalf is a

14 licensed psychiatric technician with a degree in international

15 studies.   (Metcalf Affidavit at p. 1).

16   Defendants assert that because the definitional work for the

17 R-11 Survey was undertaken by lawyers and an unqualified

18 consultant, the survey was not designed in accordance with a

19 standard scientific methodology.   According to defendants, there

20 was no design protocol or blueprint prepared **before** the R-11

21 Survey began.   They say this is borne out by the "R-11 Health

22 Study Design Protocol," attached to Clapp's 1995 report, in which

23 Clapp uses the **past tense** to describe how "towns **became**

24 disqualified as a source of control group subjects, when the

25 **interview process revealed** significant numbers of subjects

26 reporting significant radiation exposure."   (Clapp 1995 Rpt. at

27 p. 8)(Emphasis added).   This is found in the "Control Groups"

28

ORDER RE SUMMARY JUDGMENT-   248

section of the protocol.

Plaintiffs say this section of the protocol was added "after the fact" to explain why the control towns were not utilized, "even though the protocol for asking the questions (and the questions themselves) were all established in advance." In their reply brief, however, defendants cite additional text from the protocol which compellingly indicates the protocol was drafted after the survey was already underway: 1) "Lists of known graduates were **compared and verified** using yearbooks, graduating class pictures, alumni lists when available . . . ." (Clapp 1995 Rpt. at p. 7)(Emphasis added); 2) "Very few people refuse the request for authorization to obtain confirming medical records." (Id. at p. 8); and 3) "Only rarely has an interviewee asked a question related to litigation." (Id. at p. 9). Yet another example is found in "The Interview Process" section of the protocol: "Answers to questions on the survey instrument **were** in narrative and yes/no format. Codes **were** used to identify the entry of certain specifics, in certain instances." (Id. at p. 8)(Emphasis added).

Clapp's affidavit is conspicuously devoid of any response to defendants' assertion that his protocol was drafted **after** the survey was already underway. In his affidavit, he refers to "a default option built into [his] original written protocol for [his] analysis of the R-11 Survey **before** it was revealed that the original control towns had been contaminated." (Clapp Affidavit at p. 3). The court cannot find where this option is referred to in the protocol attached to the 1995 report. Furthermore,

ORDER RE SUMMARY JUDGMENT-   249

1 Clapp's deposition testimony reveals he did not recommend the

2 national comparison alternative (aka the "default option") until

3 **after** he "understood" there was a question whether the control

4 group could be used because of potential exposure.   (Clapp Dep.

5 at pp. 35-36).   This is contrary to the statement found in

6 Clapp's affidavit.

7   Defendants contend plaintiffs have provided no documentation

8 as to how the study or control group high schools were chosen.

9 They note that neither Clapp's protocol or his report provides

10 the names of the control towns and that during his deposition,

11 Clapp was unable to recall the location of the control group

12 communities.   (Clapp Dep. at p. 138).   According to defendants,

13 Metcalf failed to provide documentation of the method for

14 identifying potential survey participants from the selected

15 schools, tracking the students who attended the target schools,

16 and failed to provide response and refusal rates for the study

17 and control groups.   Therefore, defendants say there is no way to

18 assess how successful the R-11 interviewers were in identifying,

19 locating, and obtaining data from potential survey subjects, nor

20 is it possible to fully assess the bias which may have been

21 introduced as a result of the identification and location

22 process.

23   Defendants cite to portions of Clapp's deposition testimony

24 as supporting these arguments.   Clapp testified in general that

25 the response and refusal rate should be monitored for both the

26 study and control group (Clapp Dep. at pp. 18-20), but he did not

27 say Metcalf failed to do this with regard to the R-11 Survey.

28 **ORDER RE SUMMARY JUDGMENT-   250**

1 Furthermore, Clapp did not testify Metcalf failed to provide

2 documentation of the method of identifying potential survey

3 participants. Rather, Clapp discussed the necessity for defining

4 the target and control groups at the outset of the study. (<u>Id</u>.

5 at p. 15). According to Clapp, he recalls talking to Metcalf in

6 general about how to track people. He stated it was his

7 understanding Metcalf was responsible for determining how to

8 locate and track study subjects, but that a group of people,

9 including Dr. Cummins, were involved in determining the

10 eligibility criteria (i.e. which communities were considered to

11 be downwind). (<u>Id</u>. at pp. 94-96). Clapp referred to the tracing

12 procedures described in his protocol and suggested Metcalf be

13 consulted as to "paced" letters and the search and cross-

14 referencing methods employed. (<u>Id</u>. at 96-97).

15   Plaintiffs contend all of the pertinent information about

16 the **control** group communities are contained on data disks which

17 have been provided to defense counsel. They point to Metcalf's

18 affidavit as detailing the selection criteria for the survey

19 **subjects:**

20   The survey subjects were high school graduates
            from 1950-69 inclusive, from high schools in
21   14 different communities in the downwind areas
            of Washington, Oregon and Idaho.
22
            Potential participating high school graduates
23   were identified by using the school records of
            graduates, yearbooks, graduating class pictures
24   and alumni lists. Subjects were then located by
            using alumni lists when available and ultimately
25   by the extensive cross-referencing of siblings
            that is part of the interview process.
26
    (Metcalf Affidavit at pp. 2-3). A similar description is
27
28 **ORDER RE SUMMARY JUDGMENT-**   **251**

1   contained in Clapp's protocol under the heading "Locating Study

2   Subjects."   (Clapp 1995 Rpt. at p. 7).

3   Plaintiffs note the interview questionnaire used by Metcalf

4   (Foulds Ex. 92) included a field to indicate whether the

5   interview was refused.   Therefore, say plaintiffs, there is no

6   merit to defendants' claim she did not track response and refusal

7   rates.

8   Selection criteria are very important.   According to the

9   "Reference Guide on Epidemiology:"

10   A list of criteria for inclusion in and exclusion
              from the study must be articulated by the researcher.
11   These criteria should be documented clearly before
              the subjects are recruited for the study to ensure
12   that no overt or covert biases enter into the selection
              process.
13
     ("Reference Guide on Epidemiology" at p. 138).   It is apparent
14
     that Clapp does not know much about the selection criteria
15
     employed in the R-11 Survey, referring to Metcalf for information
16
     about the tracing criteria, and referring to Dr. Cummins for
17
     information about the eligibility criteria.   Metcalf is not an
18
     epidemiologist and her affidavit recites in only very general
19
     terms the selection criteria employed.
20
          Defendants cite In re TMI Litigation Cases Consol. II, 922
21
     F.Supp. 1038 (M.D. Pa. 1996), in which the district court
22
     excluded as unreliable (pursuant to FRE 702) a proffered
23
     epidemiological analysis involving a cohort study.   The
24
     epidemiologist's report did not include a discussion of study
25
     design and in his deposition, he acknowledged having no role in
26
     the conduct of the study until he received the data from two
27
28
     ORDER RE SUMMARY JUDGMENT-   252

1   other individuals who had selected the groups. The

2   epidemiologist simply performed the statistical calculations once

3   the data was provided. The two individuals who provided the data

4   were not epidemiologists, nor experts in any other scientific

5   discipline. They were not listed as experts and did not supply

6   any expert reports for the case. Id. at 1047.

7   The court found this presented a couple of problems. First,

8   it had no record evidence from which to make any judgment about

9   the qualifications of the two individuals who provided the data,

10   to create and execute the selection portion of the

11   epidemiological study design. Furthermore, because there was no

12   evidence describing the selection criteria, the defendants were

13   not able to cross-examine on this "important" issue. Thus,

14   defendants had no opportunity to influence the amount of weight

15   the jury might accord the study because there was no record

16   evidence from which they could cull their cross-examination of

17   the epidemiologist regarding the selection criteria. In

18   addition, the lack of a clearly articulated selection criteria

19   for the statistical analysis subjected the results to an enormous

20   potential rate of error. Id. at 1048.

21   The defendants in this case assert a similar situation

22   exists here in that Clapp was not involved in the design of the

23   study. Furthermore, his involvement was minimal until he

24   received data from other individuals, after which he performed

25   his statistical calculations. With the exception of the

26   mysterious Dr. Cummins, whose role in the design of the study is

27   not at all clear, none of the individuals involved in data

28   **ORDER RE SUMMARY JUDGMENT-   253**

1 collection or study design were epidemiologists or experts in any

2 scientific discipline.[192]

3   In this case, unlike TMI, there is some record evidence of

4 the selection criteria employed (Metcalf's affidavit; Clapp's

5 report), although it is severely limited in terms of detail.

6 Because of the sparse information supplied by plaintiffs

7 regarding the selection criteria, defendants legitimately argue

8 that, similar to the situation in TMI, they will not be able to

9 engage in an effective cross-examination at trial regarding those

10 criteria.

11

12   **(2)   Interview Process**

13   **(a)   Correspondence Sent to Survey Participants**

14   Defendants contend the R-11 study is flawed because contacts

15 with survey participants were "highly suggestive and injected

16 significant bias into the survey." They point to the

17 introductory letter sent by Metcalf to potential R-11 survey

18 participants, a portion of which states as follows:

19   [W]e seek . . . information concerning a
           possible relationship between the radiation
20   from nuclear facilities around the country
           and the occurrence rate of certain radiogenic
21   diseases, such as cancers, leukemia, brain
           tumors and thyroid problems. . . . We will be
22   concerned . . . if you have suffered from

23 ─────────────────────────

       [192] In her affidavit, Metcalf describes her staff as
24 including several individuals "educated in health related
   fields." Metcalf indicates that a Michele Stenehjem-Gerber,
25 Ph.D., was responsible for locating the study towns. Metcalf
   does not indicate that Stenehjem-Gerber has a background in any
26 scientific discipline. (Metcalf Affidavit at pp. 1 and 2).
   According to defendants, Stenehjem-Gerber's Ph.D. is in history.

27
28 **ORDER RE SUMMARY JUDGMENT-   254**

> any of the major diseases that can be caused
> by radiation. . . . **It is already well known
> that a great amount of lethal and damaging
> radiation has been released over the years
> into areas downwind from several nuclear
> facilities in the United States.** What is not
> known is the extent of the injury and disease
> caused by these radioactive releases.

(Metcalf Letter, Defendants' Ex. 80)(Emphasis added).

According to defendants, the letter informed potential survey participants that the study was of persons exposed to radioactive emissions from the Hanford plant[193]; that the Hanford plant had released a "great amount of lethal and damaging radiation;" the "lethal" radiation had caused untold health effects, the extent of which the survey was trying to determine; and among the health effects caused by the radiation were "cancers, leukemia, brain tumors and thyroid problems." Therefore, defendants say the letter prompted R-11 Survey participants to report thyroid problems, cancers, leukemia and brain tumors.

At his deposition, Clapp acknowledged he did not review the letter before it was sent. (Clapp Dep. at p. 125). Clapp expressed concern about bias in the letter:

> The sentence where radiogenic diseases
> such as cancers, leukemia, brain tumors
> appear in the same sentence makes me
> concerned that that is a- I don't know
> how to put it- that it leads the potential
> respondent in a certain direction.

(Id. at pp. 127-28). Clapp added, however, that his concern was diminished because of the availability of medical records to

---

[193] Indeed, the heading on the letter states: **"The R-11 Survey (A Study of Radiogenic Incidence Downwind From Hanford)."**

ORDER RE SUMMARY JUDGMENT-   255

1 verify the diseases.[194]   (Id. at p. 128).

2   Defendants also cite a follow-up letter which Metcalf sent

3 to survey participants advising that "[i]n order to further

4 validate the survey results it is necessary that the medical

5 records concerning any reported **radiogenic disease, such as**

6 **thyroid problems, brain tumors or cancer, be reviewed by a**

7 **professional epidemiologist as part of the study."** (Defendants'

8 Ex. 79)(Emphasis added). Defendants consider this "prejudicial"

9 because it also tells respondents which diseases to report and

10 assumes those diseases were caused by radiation emitted from

11 Hanford. The letter states it is regarding "Survey of the

12 Incidence of Radiogenic Diseases Downwind from **Hanford."** Clapp

13 was not sure whether he had seen this letter before or after it

14 was sent out. (Clapp Dep. at p. 129).

15   In September/October 1992, Judith Jurji, president of the

16 Hanford Downwinders Coalition (HDC), sent a letter to "study

17 subjects" of the R-11 Survey. The letter begins by indicating

18 the mission of the HDC is to "provide information and support to

19 people who lived in **the pathway of the contamination from the**

20 **Hanford nuclear facilities."** It adds that HDC sponsored the R-11

21 Survey "which is an investigation concerning the possible

22 correlation between the Hanford nuclear emissions and the

23 'radiogenic' health problems experienced by some persons living

24 downwind or downriver from Hanford." Thyroid problems, cancers

25

    [194] This argument will be discussed in more detail *infra*.
26 Essentially, plaintiffs assert that confirmatory medical records
   are enough to overcome any and all flaws in the design and
27 execution of the R-11 Survey.

28
ORDER RE SUMMARY JUDGMENT-   256

1   and tumors were identified as "radiogenic" diseases. Jurji's

2   letter discussed the limitations period for filing a legal claim

3   and advised that HDC's attorney was Tom Foulds who was

4   "prosecuting a suit which began on August 1990 for claims for

5   recovery for illness or injury on behalf of approximately 1200

6   clients against the various contractors . . . that operated

7   Hanford for the government." R-11 Survey subjects were advised

8   to contact Mr. Foulds if they wanted to join the litigation. The

9   letter stated it was not recommending that anyone should or

10 should not bring a claim. (Defendants' Ex. 58).

11   Jurji's letter is clearly the most egregious of all in

12 suggesting the illnesses of study subjects were caused by Hanford

13 emissions and furthermore, in inviting them to join a lawsuit

14 against the Hanford contractors.[195]

15   The plaintiffs contend there is nothing false or misleading

16 in either of Metcalf's letters. They assert that during the

17 early 1990s the media reported "the Hanford nuclear exposures

18 coverup- the types of radiogenic diseases expected- so many of

19 the Study participants already had a very good understanding of

20 the Hanford coverup." In addition, they assert it is not

21 "prejudicial" to state that radiogenic disease can include

22 thyroid problems, brain tumors, and cancer when "it is simply a

23 medical fact." In his affidavit, Clapp contends the introductory

24 _____

25   [195] At his deposition, Clapp indicated he had not previously
    seen the Jurji letter and "[w]ho [it] went to, and you know,
26 specifically, how many of these letters went out, I have no
    idea." Clapp stated he never had any contact with Jurji or any
27 members of HDC. (Clapp Dep. at pp. 132-33).

28

ORDER RE SUMMARY JUDGMENT-   257

1   letter did not tell respondents they had been exposed to "lethal

2   and damaging" radiation.   (Clapp Affidavit at p. 3).

3   Plaintiffs miss the point about the Metcalf letters.   The

4   introductory letter does not state survey respondents were in

5   fact exposed to "lethal and damaging" radiation.   However, along

6   with the other statements and information in that letter, a

7   reasonable person could easily understand the letter to tell

8   him/her that his/her illness was caused by "lethal and damaging"

9   radiation from Hanford.   And although "thyroid problems, brain

10 tumors and cancer" may be radiogenic in origin, the point is the

11 letters tell the survey respondents what conditions they should

12 report, and that they should assume those conditions are in fact

13 radiogenic in origin.

14   Defendants persuasively argue the fact the survey

15 communities were subject to media reports of a "Hanford coverup"

16 before commencement of the survey, made it even more important

17 for plaintiffs to consider a survey design that would eliminate

18 bias.   At a minimum, it should have made the R-11 team extremely

19 vigilant to avoid communications which might influence survey

20 responses.

21   With regard to the Jurji letter, plaintiffs contend that

22 since the letter was circulated only to participants who had

23 already been surveyed, it could not have influenced their

24 responses.   The problem here, say defendants, is no steps were

25 taken to make sure the letter was not discussed with, or copies

26 circulated to, study group members who had yet to be interviewed.

27 Clapp's deposition testimony indicates interviews were still

28

ORDER RE SUMMARY JUDGMENT-   258

1 ongoing at that time.   (Clapp Dep. at p. 121).[196]   Defendants

2 say that a database of survey responses indicates interviews were

3 ongoing with thirteen of fourteen survey communities when the

4 letter was sent.   Plaintiffs do not dispute that all of the

5 interviews were not completed at the time the Jurji letter was

6 sent.

7   None of these letters were carefully thought out in terms of

8 their real potential for injecting bias into the survey.   Even

9 Clapp expressed concern about this potential.   The slanted nature

10 of the letters is obvious.

11

12   **(b)   Questionnaire**

13   According to defendants, another methodological problem is

14 the R-11 interviewers failed to use a standardized or structured

15 questionnaire in conducting the interviews.   Defendants say there

16 were no instructions setting forth the precise questions to be

17 asked, nor were there any guidelines concerning the phrasing of

18 questions.

19   At his deposition, Clapp acknowledged that a structured

20 questionnaire- one which specifically defines the questions that

21 will be asked and gives instructions to the interviewer as to how

22 to proceed depending on the response given- is desirable,

23 particularly with a telephone survey.   This is so because the

24 information is "likely to be more consistent from one interview

25 _____

26   [196]   According to Clapp, interviews were conducted over a
   three or four year period from roughly 1991 to 1994.   (Clapp Dep.
27 at pp. 124-25).

28
   ORDER RE SUMMARY JUDGMENT-   259

1 to the next," as opposed to an "open-ended" questionnaire where

2 much information is collected without answering a specific

3 question and consequently, is open to interpretation by the

4 person doing the survey.[197] Clapp acknowledged that without a

5 structured questionnaire, responses could differ depending on the

6 questions asked. He added that the use of a structured

7 questionnaire insures the standardization of data collection.

8 (Clapp Dep. at pp. 21-23).

9   Clapp was asked about the questionnaire used in the R-11

10 Survey. Clapp conceded there was no standardized or structured

11 definition of the questions to be asked during the interviews.

12 (Clapp Dep. at p. 111-12). He conceded the questionnaire

13 (Defendants' Ex. 99) did not indicate the precise manner in which

14 each of the questions was to be phrased. It lists a variety of

15 medical conditions next to which is a "Y/N" for Yes/No, "Date

16 Diagnosed" and "Age at Diagnosis." (Clapp Dep. at pp. 112-14).

17 Clapp said he did not know the exact language which was used by

18 the interviewer. (Id. at pp. 118-19).

19   Other than the questionnaire itself, Clapp was not aware of

20 any documents providing further details about the phrasing of

21 _____

22   [197] Defendants' expert Howe agrees with Clapp. In his
    affidavit, Howe states the use of a structured questionnaire,
    prescribing the exact wording of each question and providing

23 instructions on how to proceed depending on the answer, prevents
    the interviewer from deviating from a specific form of question.

24 This protects against subconscious and conscious bias on the part
    of the interviewer. Without a structured questionnaire, the

25 interviewer can manipulate the question to generate a particular
    result. (Howe Affidavit at pp. 6-7; Ex. B to Defendants' Reply).

26 This takes on added significance if, as Clapp testified, Metcalf
    knew when she was speaking to study subjects as opposed to

27 control group members. (Clapp Dep. at p. 131).

28 **ORDER RE SUMMARY JUDGMENT-   260**

questions to be used during the interview process. (Clapp Dep.
at p. 115). Although Clapp opined that the R-11 Survey Database
Protocol (p. 10 of Clapp's 1995 Rpt.) provided some guidelines
for the phrasing of questions regarding location and employment
history, he could not say the same was true for any of the other
questions. (Clapp Dep. at pp. 116-17).

Clapp testified he had assurances from Metcalf that the
questions were asked in the same manner during each interview.
Clapp also stated that because the responses in the
"overwhelming" number of cases were "verified" by medical
documentation, this provided "some assurance that questions were
asked in a way that got consistent responses." (Clapp Dep. at
pp. 117-18).

The plaintiffs cite Clapp's affidavit in which he asserts
"[t]he R-11 survey interview team used a structured questionnaire
that was not in any way open ended." (Clapp Affidavit at p. 2).
This assertion is interesting in light of Clapp's deposition
testimony in which he conceded: 1) there was no standardized or
structured definition of the questions to be asked in the
interview; 2) the questionnaire did not indicate the precise
manner in which each of the questions was to be phrased; 3) he
was not aware of any documents providing further details about
the phrasing of questions to be used during the interview
process; and 4) he advised Metcalf the questions should be asked
the same way every time and relied on her assurances that they
were so asked.

In her affidavit, Metcalf states:

ORDER RE SUMMARY JUDGMENT-   261

> Using a computer database specifically designed
> for the questions in the study, I asked the identical
> questions in the same format every time on each study
> subjects (sic).
>
> There was very little latitude to vary from the
> standard yes/no format, except in the few instances
> when the interviewee was uncertain as to their
> medical condition, in which case we recorded their
> response verbatim in the expanded field.

(Metcalf Affidavit at p. 4).

Just looking at the questionnaire used in the R-11 Survey, it is obvious, particularly with regard to the questions about medical conditions, that the interviewers had discretion as to how they phrased the questions.[198] Metcalf really does not say exactly how she asked the "yes/no" questions. Furthermore, Metcalf was not the only interviewer.

Examination of the 1994 NHIS Survey questionnaire (Defendants' Ex. 90) reveals the discretion possessed by the R-11 interviewers. The NHIS Survey interviewers were told precisely how to ask the questions pertaining to medical conditions:

> Now I am going to read a list of medical
> conditions. Tell me if anyone in the family
> has had any of these conditions, even if you
> have mentioned them before. DURING THE PAST 12
> MONTHS, did anyone in the family have- a goiter
> or other thyroid trouble?; diabetes?; etc.

The NHIS questionnaire also offers a definition of the medical conditions, unlike the R-11 Survey.

Although the defendants raise valid arguments concerning survey design and the interview process, it is not readily

---

[198] One example of the interviewers having discretion is the fact that in many cases, the field for identifying "Mr/Mrs/Ms" was not completed. (Clapp Dep. at p. 136).

ORDER RE SUMMARY JUDGMENT-   262

1 apparent how these flaws actually affected the accuracy of the

2 survey. Nevertheless, the important consideration is whether the

3 mere existence of these flaws raises a significant doubt that

4 there was some adverse impact. Plaintiffs' response is that to

5 the extent there were any flaws, they were alleviated through

6 examination of medical records verifying reported medical

7 conditions. That issue is discussed _infra_.

8

9   **(3) Dropping the Control Group**

10   The R-11 Survey started out with a control group. The

11 purpose was to compare the incidence of certain diseases in the

12 exposed downwinder communities (the study subjects) versus

13 unexposed communities. However, the control group was eventually

14 dropped. Clapp describes this in his R-11 Health Study-Design

15 Protocol:

16   Attempts to locate a control group have
           been unsuccessful. Three towns were
17   chosen for this purpose. The towns were
           located in geographic areas with micro-
18   climates and agricultural bases nearly
           identical to the primary target groups,
19   that were thought to be relatively isolated
           from exposure to radioactive elements. In
20   each case, the towns became disqualified
           as a source of control group subjects, when
21   the interview process revealed **significant**
           numbers of subjects reporting **significant**
22   radiation exposure.

23 (Clapp 1995 Rpt. at p. 8)(Emphasis added).

24   According to defendants, the real reason the control groups

25 were dropped is because the downwinder respondents (study

26 subjects) reported less **thyroid cancer** than the abandoned control

27 group, and the two groups reported essentially the same level of

28 **ORDER RE SUMMARY JUDGMENT-   263**

1 thyroid nodules. Defendants say the decision to discard the

2 control group is "an unacceptable departure from standard

3 scientific methodology and completely negates the reported

4 results of the survey."

5   At his deposition, Clapp was unable to name the control

6 group communities. He did not know how they were chosen. He

7 testified the control group was discarded because it was his

8 **"understanding** from Miss Metcalf primarily and also from Mr.

9 Foulds . . . that there turned out to be numerous examples of . .

10 . radioactive material exposure in the people in the control

11 towns. . . ." (Clapp Dep. at pp. 137-38)(Emphasis added). Clapp

12 acknowledged the decision to discard the control group occurred

13 during the middle of the interview process. According to Clapp,

14 the decision was his "in consultation with Miss Metcalf and with

15 Mr. Foulds." (Id. at pp. 139-40).

16   Clapp testified he has disks in his possession which contain

17 information about the control groups, however he has not reviewed

18 the information. Otherwise, Clapp stated he knew nothing about

19 the control groups. (Id. at pp. 150-51). Clapp testified he had

20 not looked at the disks because he "was focusing on things he

21 needed to do in order to produce [his] reports and [he] was not

22 going to analyze the control area data although . . . potentially

23 it might be of interest to look at it some day if somebody wants

24 to pay for that . . . ." (Id. at p. 161).

25   Defendants argue the decision to disregard control group

26 data cannot be justified by Clapp's refusal to apprise himself of

27 the facts underlying the decision. This is so, they say, because

28 **ORDER RE SUMMARY JUDGMENT-   264**

1   Clapp admits that relevant to the propriety of dropping a control

2   group is knowing whether the interviews suggested a higher

3   prevalence of disease in the control group than in the study

4   group. (Clapp Dep. at pp. 155-56). Furthermore, defendants

5   point out that even if Clapp was ignorant of the results of the

6   control group interviews, Metcalf and Foulds were not.

7   In their initial brief, the defendants do not explain how

8   they arrived at the conclusion there was a higher prevalence of

9   thyroid cancer among the control group communities and about the

10 same prevalence of thyroid nodules. In their reply brief,

11 defendants offer the affidavit of their expert, Dr. Howe, who

12 says that control area interviews revealed a thyroid cancer

13 prevalence of .38% (4 out of 1052) as compared to the lower study

14 group thyroid cancer prevalence reported by Dr. Clapp of .30% (22

15 out of 7366). Apparently, Dr. Howe gleaned this information from

16 his review of the computer disks supplied by defendants. Howe

17 says that at the time it was decided to discard the control

18 group, over 1,000 interviews had already been conducted with

19 control group members. (Howe Affidavit at p. 11, Ex. B to

20 Defendants' Reply).

21   In their response brief, plaintiffs refer specifically to

22 the control group town of Oroville, Washington and assert

23 "[d]efendants' allegation (without any supporting reference) that

24 the control group town of Oroville was dropped because it showed

25 a higher rate of illness than the Study towns **is false**." This is

26 somewhat confusing in that it is not apparent in defendants'

27 opening brief or their reply brief that they refer specifically

28 **ORDER RE SUMMARY JUDGMENT-   265**

1   to Oroville, as opposed to the control group communities as a

2   whole- Oroville; Basin, Wyoming; and Vega, Texas.

3   According to plaintiffs, Oroville was included as a control

4   town under the belief its location would take it out of Hanford's

5   I-131 air dispersion pathway. However, plaintiffs say they

6   subsequently learned Oroville was on the boundary of the exposure

7   area when the final report on HEDR (Hanford Environmental Dose

8   Reconstruction Project) was released in 1994. Plaintiffs say

9   Oroville was dropped after 455 interviews had been completed, not

10 over 1,000 as claimed by defendants. Plaintiffs assert that at

11 the time Oroville was dropped, the prevalence rate of **"thyroid**

12 **illness"** was still "significantly less" than the exposed study

13 towns, and remained that way through the completion of the study.

14   Howe's claim that the control group was discarded after

15 1,000 interviews appears to be based on the **total** number of

16 interviews conducted between all three control group towns, not

17 just Oroville. Indeed, in their Motion to Strike materials,

18 plaintiffs indicate that 80% of the target level interviews were

19 completed for both Basin, Wyoming and Vega, Texas.[199] Assuming

20 defendants' argument of a higher prevalence of thyroid cancer in

21 the control group is based on an examination of the data from all

22 three control group communities, not just Oroville, nowhere do

23 plaintiffs (even in their Motion to Strike) specifically take

24 issue with that argument or Howe's figure of .38% (4 out of

25 _____

26   [199]   In the chart attached to their reply brief, defendants
    assert there was a total of 1,157 interviews from the three
27 initially selected control towns.

28 **ORDER RE SUMMARY JUDGMENT-   266**

1   1052).   If only 455 interviews were conducted with the Oroville

2   group, as asserted by plaintiffs, the other 600 interviews must

3   have been with the Basin and Vega groups.   The court notes also

4   that plaintiffs refer to "thyroid illness," not specifically to

5   thyroid cancer.   "Thyroid illness" is a general term which can

6   include non-cancerous diseases.   Defendants specifically argue

7   the prevalence of **thyroid cancer** was higher in the control group

8   communities than in the study group communities.[200]

9   The plaintiffs essentially argue the control group data is

10   simply irrelevant since Clapp did not utilize it.   Plaintiffs

11   also argue that defendants do not challenge the fact control

12   towns were exposed or that controls should not be contaminated.

13   Plaintiffs badly miss defendants' point which is that in order to

14   determine **whether or not** he should have utilized the control

15   group data, Clapp, as a professional epidemiologist, should have

16   at least reviewed the data in order to make an intelligent

17   _____

18   [200]   Again, in their Motion to Strike, plaintiffs assert that
     the control towns "all show a significantly lower prevalence of
19   **thyroid problems** than the Hanford downwinder study groups . . .
     ."   (Plaintiffs' Motion at p. 28, n. 3).   "Thyroid problems"
20   encompass both neoplastic and non-neoplastic diseases.
     Defendants contend the prevalence rate for **thyroid cancer** was
21   higher in the control towns than the study towns.
          Elsewhere in their Motion to Strike, plaintiffs make an
22   argument which appears to reveal their awareness of this critical
     distinction.   Plaintiffs say "[i]f defendants['] estimate of
23   thyroid cancer in the original control group is accurate, this
     supports the belief that they were exposed and hence **thyroid
24   cancer** prevalence is not surprising."   (Plaintiffs' Motion at p.
     35).   In other words, plaintiffs seem willing to agree there is
25   an increased thyroid cancer prevalence in the control towns if
     that proves the control towns were indeed exposed to the extent
26   it was justifiable to drop them.   Otherwise, they are not willing
     to concede there is an increased prevalence.   The plaintiffs are
27   evasive on this point.

28   **ORDER RE SUMMARY JUDGMENT-   267**

1   assessment regarding utilization thereof. And rather than just

2   taking the word of non-experts that the control group towns were

3   contaminated, Clapp should have satisfied himself that was the

4   case before agreeing to discard the control group.

5   As noted above, Clapp testified it was his "understanding"

6   from Miss Metcalf and Mr. Foulds that there were "numerous"

7   examples of radioactive exposure in the control towns. Clapp

8   said he "believe[d]" the exposure was from other nuclear

9   facilities and potentially some from Hanford which "had traveled

10 in directions that were not previously understood." (Clapp Dep.

11 at pp. 138-39). Clapp simply did not know what purportedly

12 constituted the "significant numbers of subjects" and

13 "significant exposure" he referred to in his written protocol as

14 justification for dropping the control towns.

15   Even assuming the control towns were contaminated, the fact

16 of contamination is further evidence the survey was not properly

17 designed. The R-11 Survey team chose three control towns and in

18 each case failed to assess the possibility of contamination.

19 This is additional confirmation that there was no guidance from a

20 professional epidemiologist when the survey was being put

21 together.

22

23   **(4)   Use of Substitute Comparison Groups**

24   **(a)   NHIS Survey**

25   After dropping the control towns, plaintiffs needed to

26 substitute a new set of control data and turned to the National

27 Health Interview Survey (NHIS) of the U.S. Census Bureau.

28 **ORDER RE SUMMARY JUDGMENT-   268**

1 Defendants claim the data from the NHIS Survey is not comparable

2 to the R-11 Survey.

3   According to defendants, one significant difference is the

4 surveys posed different questions to survey participants.

5 Whereas the R-11 Survey asked about lifetime occurrence of

6 thyroid disease, the NHIS Survey asked about the occurrence of

7 thyroid disease within the past year. In his 1995 Report, Clapp

8 stated the "R-11 Survey Rate for Goiter and Other Disorders of

9 the Thyroid may not be directly comparable to the U.S. Rate

10 because the National Health Interview Survey did not ask

11 **precisely the same question."** (Clapp 1995 Rpt. at p. 6)(Emphasis

12 added).

13   The plaintiffs consulted Harold Javitz, Ph.D., of the

14 Stanford Research Institute, asking him to comment on how

15 differences between the R-11 Survey and the NHIS Survey could

16 affect estimates of the prevalence of thyroid and goiter

17 conditions. Dr. Javitz noted the R-11 Survey asked about "ever"

18 having thyroid disease and the NHIS asked about having thyroid

19 disease "in the past 12 months." (Foulds Ex. 63). Furthermore,

20 Javitz pointed out that the R-11 Survey asked about thyroid

21 problems using six descriptors ("thyroid cancer, hypothyroidism,

22 hyperthyroidism, thyroid nodules, goiter or other thyroid

23 problems"), whereas the NHIS Survey asked about thyroid problems

24 using two descriptors ("a goiter or other thyroid trouble").

25   Javitz indicated that both of these factors would tend to

26 increase the prevalence of reported cases in the R-11 Survey

27 versus the NHIS Survey. Persons asked whether they "ever" had a

28 **ORDER RE SUMMARY JUDGMENT-   269**

1 disease would answer more often in the affirmative than persons

2 asked if they had the disease within the last twelve months.

3 Persons asked about thyroid problems using a variety of different

4 descriptors (as in the R-11 Survey) would have a greater tendency

5 to remember thyroid problems than someone given fewer

6 descriptors. (Ex. 63 at pp. 3-4).

7   Defendants claim the NHIS data, unlike the R-11 Survey data,

8 cannot be adjusted for gender because the R-11 interviewers

9 failed to collect gender information. Clapp acknowledged the R-

10 11 statistical analysis combined the average rates for men and

11 women. Clapp stated he was unaware of any published studies

12 dealing with thyroid conditions that do **not** account for gender.

13 He acknowledged the difference in thyroid condition prevalence as

14 between women (higher) and men (lower), but did not know for

15 certain the breakdown of men and women in the R-11 Survey.

16 (Clapp Dep. at pp. 134-36).

17   In his report, Dr. Javitz states gender breakdown is

18 definitely material and could well make the NHIS prevalence rate

19 higher versus the R-11 rate. For example, if older females

20 outnumbered older males in the R-11 Survey by 60% to 40%, then

21 the NHIS prevalence rate in the 45 to 64 age category should be

22 increased. Javitz emphasized "[t]he substantial difference

23 between the gender-specific prevalence rates should be taken into

24 account," and this was not done in the comparison of the R-11

25

26

27

28 **ORDER RE SUMMARY JUDGMENT-   270**

1 Survey prevalence versus the NHIS prevalence.   (Ex. 63 at p.

2 5).[201]

3   In the NHIS Survey, there was an 18-44 age category. Clapp

4 used that category for comparison purposes, **even though the R-11**

5 **data did not include individuals as young as 18.** (Clapp 1995

6 Rpt. at p. 6). Participants in the R-11 Survey were individuals

7 who graduated from high school 20 or more years ago. Therefore,

8 the youngest participants in the R-11 Survey were 38 years old.

9 Accordingly, Clapp's comparison group from the R-11 Survey was 38

10 to 44 year olds. Dr. Javitz explained how this could account for

11 a higher prevalence rate in the R-11 Survey versus the NHIS

12 Survey:

13   The prevalence rate of thyroid and goiter
            conditions **increases substantially with age.**
14   Unfortunately, the younger age category for
            the R-11 and the NHIS Survey **may not be**
15   **completely comparable.** Nearly all members
            in the R-11 Survey who are in the 18 to 44
16   age category will be 39 years or **older.** A
            large percentage of the NHIS respondents who
17   are in the 18 to 44 age category will be
            younger than 39. Thus, we would expect a some-
18   what **lower** prevalence rate in the NHIS relative
            to the R-11 Survey who are in the 18 to 44
19   year age category because of different age
            distributions.
20
   (Ex. 63 at p. 6)(Emphasis added).
21
        Defendants contend Clapp failed to consider and adjust for
22
   potential confounding factors or population characteristics
23

24 _____

        [201] Plaintiffs claim they could have accounted for gender
25 differences if they wanted to because all they had to do was look
   at the names of the interviewees. However, as defendants point
26 out, names are not always an accurate indicator of gender,
   particularly with cross-gender names such as Chris, Pat, Lee,
27 etc.
28
   ORDER RE SUMMARY JUDGMENT-   271

1 distinguishing the R-11 respondents from the national survey

2 respondents.   Because the R-11 subjects were all high school

3 graduates, defendants assert they would have better access to

4 health care facilities than a general population sample, leading

5 to higher diagnosis rates of thyroid disease in the R-11 group

6 compared to the national group.   Indeed, in his affidavit, Clapp

7 acknowledges health care access affects response rates and "was a

8 factor in our survey."   According to Clapp if R-11 Survey

9 participants "had not had access to health care[,] we would not

10 have been able to do the critical step of reviewing medical

11 records to confirm their reports of illness."   (Clapp Affidavit

12 at p. 4).   Although Clapp says health care access was a factor,

13 he does not say this factor was taken into account in comparing

14 R-11 and national prevalence rates.   All he says is that health

15 care access made it possible to confirm the illnesses reported by

16 R-11 respondents.

17   Citing the affidavit of Dr. Sara Peters (Foulds Ex. 89),

18 defendants note that areas downwind of Hanford were goitrogenic

19 (iodine deficient) prior to and at the time of the radiation

20 releases.   This, say defendants, also distinguishes the R-11

21 group from the national group because it presumably could result

22 in the R-11 group reporting more cases of thyroid disease.   This

23 argument was not specifically presented in defendants' opening

24 brief on the R-11 motion in limine.   Therefore, plaintiffs do not

25 address it in their response brief.   However, defendants have

26 brought the issue up in their other motions in limine and it is a

27 salient point.   Furthermore, the court notes that in their

28 **ORDER RE SUMMARY JUDGMENT-   272**

1 response brief, the plaintiffs invited defendants to "raise any

2 possibility of missed confounding factors in their reply."

3 (Plaintiffs' Response Br. at p. 17).

4   The plaintiffs say that although Dr. Javitz identifies a

5 number of factors which could **increase the R-11 prevalence rate**

6 **versus the national survey rate**, he also identifies a number of

7 factors which could **increase the national survey rate versus the**

8 **R-11 rate.** Essentially, Javitz opines the factors balance out:

> . . . it appears reasonable to me to compare
> the R-11 survey prevalences of thyroid and goitre
> (sic) problems to the NHIS derived prevalences.
> Although the surveys are not identical, they
> appear to be comparable and similar in important
> ways. Both surveys are based on discussions
> with interviewers and both elicit responses
> about chronic conditions from checklists. There
> are some factors which would act to increase the
> rate in the R-11 survey relative to the NHIS
> (e.g. time period of condition, specificity of
> description of the condition, age of respondents
> in the 18 to 44 year category and possibly sex
> of the respondent), other factors which would
> act to increase the rate in the NHIS relative to
> the R-11 Survey (e.g. lead-ins to diseases, method
> for counting refusals to participate, and in-person
> interviews), one factor that decreases the effect of
> time period of condition on increasing the rate in the
> R-11 survey relative to the NHIS (e.g. greater
> underreporting for conditions that were not active in
> the last year), and a factor that tends to support
> the conclusion of a higher rate of thyroid and
> goiter conditions among the R-11 population (e.g. the
> higher confirmation rate in the R-11 survey relative
> to the SRI survey).[202] **In my opinion, consideration**

---

[202] Javitz refers to a Stanford Research Institute (SRI)
study using "NHIS-like questions" to ask whether patients had any
physician visits in the last 12 months for thyroid trouble or
goiter. Of those who responded affirmatively, 58.1% of the
diagnoses were confirmed by medical records. Patients were also
asked whether they had malignant neoplasms (in any body location)
that resulted in a physician visit within the last 12 months. Of
those who responded affirmatively, 55.7% of the diagnoses were
confirmed by medical records. Javitz compared these confirmation

ORDER RE SUMMARY JUDGMENT-   273

**of these factors would not change the conclusion of
a substantially higher rate of thyroid and [goiter]
problems among the R-11 population than among the
NHIS population.**

(Ex. 63 at pp. 8-9) (Emphasis added).

Defendants contend Javitz does not independently validate
Clapp's methodology, nor does he state Clapp's comparison of the
R-11 and NHIS surveys is consistent with standard epidemiological
methodology.

The Javitz report is an "after the fact" analysis. Clapp
had already made the comparison between the R-11 Survey and the
NHIS Survey. Javitz does not opine the comparison was a
methodologically sound idea in the first place. Indeed,
considering the number of factors Javitz identifies as
potentially affecting the prevalence rates between the two
surveys, one must question whether he would have considered it
methodologically sound to embark upon the comparison (knowing one
of the age categories was not completely comparable; knowing
there may not have been completely accurate gender information
from the R-11 group, etc.) Javitz's report is offered more as
support for Clapp's **ultimate conclusion**, as opposed to his
methodology.

Furthermore, Javitz' opinion is confined to the comparison
between the two surveys. He offers no opinion about anything
which occurred prior to that time, specifically with regard to
the manner in which the R-11 data was generated (i.e. letters

_____

rates to what he reported as an 81.1% confirmation rate in the R-
11 Survey for "thyroid problems" (non-neoplastic) and a 91.7%
confirmation rate for thyroid cancers. (Ex. 63 at p. 6).

ORDER RE SUMMARY JUDGMENT-   274

1   sent to R-11 Survey participants, the questionnaire used, the

2   dropping of the control group, etc.).[203] The comparison of

3   surveys is ultimately irrelevant. The R-11 data was so badly

4   tainted because of methodological flaws in the collection process

5   that, as a threshold matter, it was improper to even embark upon

6   the comparison.

7

8   **(b) Connecticut Tumor Registry**

9   Defendants argue the Connecticut Tumor Registry is also not

10   an appropriate comparison group for determining prevalence of

11   **thyroid cancer** among the R-11 group. Clapp used prevalence rates

12   taken from a journal article based on the Connecticut Tumor

13   Registry data. (Feldman, et al., "The Prevalence of Cancer:

14   Estimates Based on the Connecticut Tumor Registry," 315 **The New**

15   **England Journal of Medicine** 1394-1397 (November 27, 1986)). He

16   used the prevalence rates for males and females in age groups 30-

17   49 and 50-59 and came up with a "combined" prevalence rate by

18   averaging the sex-specific rates:

19

Age Group	Female	Male	Average
30-49 years	112.7/100,000	32.4/100,000	72.6/100,000
50-59 years	146.6/100,000	52.9/100,000	99.8/100,000

24   According to defendants, Clapp calculated a combined average

25   _____

26   [203] Javitz explicitly acknowledged as much in a November 13,
     1997 memorandum he sent to plaintiffs' counsel. (Ex. D to
27   Evenson Plaintiffs' Motion to Strike materials; Foulds Ex. 306).

28   **ORDER RE SUMMARY JUDGMENT-   275**

1 for both sexes for each of these groups, "implicitly assuming the

2 R-11 Survey group was evenly divided among males and females."

3 Indeed, in Clapp's 1996 supplemental report, it is not evident

4 that he offers a breakdown of the reported thyroid cancer cases

5 by gender. Although he may not have considered it necessary to

6 do so, there is some question, as noted above, whether he would

7 have been able to do it accurately since specific gender

8 information was not recorded for the R-11 survey

9 participants.[204] Furthermore, the 30-49 age category is not

10 completely comparable in that the R-11 survey participants were

11 all apparently at least 38 years old. The 30-49 age category

12 derived from the Connecticut data would have included some

13 individuals younger than those in the R-11 group. The parties

14 agree the risk of thyroid cancer diminishes with advancing age.

15   Clapp used his average rates (derived from the Connecticut

16 data) and multiplied it by the number of R-11 subjects in each

17 age group to predict the number of cancers expected among the R-

18 11 group:

Age Group	Average Rate		No. of R-11 Subjects		Expected No. of Cancers
30-49	.0726%	x	4582	=	3.3
50-59	.0998%	x	2784	=	2.8
		TOTAL	7366		6.1

---

[204] There is a consensus among the experts that age and sex are the greatest modifiers of thyroid cancer risk.

ORDER RE SUMMARY JUDGMENT-   276

1 Although defendants allude to what they believe is the

2 methodological impropriety of comparing the R-11 data and the

3 Connecticut data (because of the gender and age breakdown used by

4 the Connecticut data, discussed supra), their primary argument is

5 the thyroid cancer prevalence among the R-11 study subjects

6 should have been compared to the prevalence in the R-11 control

7 group. As discussed above, it appears the prevalence rate for

8 **thyroid cancer** was higher among the control group than the study

9 group. Defendants argue there was no valid reason for dropping

10 the control group (discussed supra).

11 Here again, the R-11 data was so badly tainted because of

12 methodological flaws in the collection process (i.e. dropping the

13 control group) that comparison of surveys becomes irrelevant. As

14 with the NHIS survey, it was improper to even embark upon any

15 comparison with the Connecticut data. It is irrelevant whether

16 there were any methodological flaws in making the comparison.

17

18 **(5) Medical Record Confirmation**

19 Richard Bird, M.D., also of JSI Center for Environmental

20 Health Studies, was responsible for verifying reported cases of

21 thyroid disease through review of medical records of R-11 survey

22 participants. According to Clapp's 1995 report:

23 > In reviewing medical records for the R-11 Survey,
> Dr. Bird focused on identifying symptoms, physical
24 > exam signs, laboratory or radiologic findings, and
> physician assessment and treatment methods that
25 > were consistent with various types of thyroid
> diseases and cancers. His approach to each
26 > record submitted, was to base his conclusion
> on the information provided in the records
27 > submitted. At the end of his review of each
28

ORDER RE SUMMARY JUDGMENT-   277

record, he summarized his conclusion and, in
doing this, considered the disease condition(s)
that were reported by the patient during the
survey interview process. He commented on these
self-reported diagnoses, so that his conclusion
reflected whether the medical record submitted
had been obtained for the correct diagnosis
and whether there were records from a different
period of time that might contribute further to
understanding the diagnoses. Dr. Bird considered,
in this review, the fact that physicians vary in
the degree of thoroughness with which they document
disease conditions in their records, and in each
case Dr. Bird listed the dates and the portion
of the medical records from which he drew his
conclusion.

(Clapp 1995 Rpt. at p. 2).

Plaintiffs claim Dr. Bird's record review and confirmation

of diagnoses makes up for any concerns about how the survey was

conducted (i.e. the possibility of bias). Defendants contend

that is not true. First, they claim Bird did not "confirm" the

majority of self-reported cases. Defendants note that in his

1995 report, Clapp indicated Bird had reviewed medical records

for 444 of the self-reported thyroid disease cases, which is less

than 40 percent of the total number of cases (approximately

1150). (Clapp 1995 Rpt. at p. 3).

In their Motion to Strike materials, the plaintiffs say the

medical record review is an "ongoing process that has now

surpassed a majority level (50%) and is still climbing; most

importantly these records verify the reported cancers at a rate

of 99%." (Citing Metcalf Affidavit, Foulds Ex. 222, and Bird

Letter, Foulds Ex. 144). The fact remains, however, there are a

substantial number of self-reported diagnoses which need to be

confirmed.

ORDER RE SUMMARY JUDGMENT-   278

1   Defendants contend Bird had no written protocol to guide his

2   decision as to whether medical information confirmed a reported

3   condition. According to defendants, despite their requests for a

4   protocol, none was supplied to them. In addition, defendants

5   contend Bird's summaries reveal he did not apply any uniform or

6   standard confirmation criteria. According to defendants, cases

7   Bird categorizes as "confirmed" are, in fact, unconfirmed. As

8   examples, the defendants offer some of Bird's summaries of

9   hypothyroidism cases which he treated as "confirmed" cases.

10   Defendants assert the notes of the treating physician in each of

11   these cases show that hypothyroidism could not be "confirmed."

12   (Defendants' Reply Br. at p. 21).

13   Defendants argue that it is of even more significance that

14   Clapp did not use "confirmation" adjusted numbers in arriving at

15   his prevalence rates, but instead used "self-reported cases of

16   thyroid disease" (along with the confirmed cases).   Indeed, in

17   his 1995 report, Clapp states that "[i]n order to have data that

18   were comparable to the NHIS data, we chose to analyze **self-**

19   **reported cases of thyroid disease** in the results presented . . .

20   ." (Clapp Rpt. at p. 5). Defendants cite deposition testimony

21   from Clapp confirming he used self-reported cases (unconfirmed)

22   as well as confirmed cases (in his analysis of the prevalence of

23   non-neoplastic thyroid disease). (Clapp Dep. at pp. 167-69; 266-

24   67).

25   Plaintiffs contend these arguments are inappropriate because

26   defendants did not raise them in their opening brief when they

27   should have. Dr. Bird's summaries (and the accompanying medical

28   **ORDER RE SUMMARY JUDGMENT-   279**

1   records) were the subject of a discovery dispute which was not

2   resolved until late August 1997, after the defendants submitted

3   their opening iodine brief and plaintiffs submitted their

4   response brief. Discovery Master Johnson ordered plaintiffs to

5   produce the summaries and the records. According to plaintiffs,

6   they were willing all along to supply the summaries and records,

7   provided defendants executed a confidentiality agreement to

8   preserve the privacy of the survey participants. Plaintiffs say

9   defendants did not, or were not willing, to provide the

10   confidentiality agreement until after reading the plaintiffs'

11   response brief (which, as noted above, contends Bird's

12   confirmation of reported diseases verifies the accuracy of the R-

13   11 Survey). At that point, plaintiffs say they brought the

14   matter before the discovery master because of concern the

15   defendants would use the summaries in their reply brief, as they

16   indeed have done. It is for this reason, plaintiffs contend

17   defendants' arguments premised on Bird's summaries should be

18   stricken.

19   In their Motion to Strike materials (effectively a surreply

20   in many respects), the plaintiffs assert it is "misleading" on

21   the part of defendants to argue Bird did not apply any uniform or

22   written criteria. They claim such criteria are set forth in

23   Clapp's April 1996 report, however it appears they actually mean

24   Clapp's 1995 report (Clapp 1995 Rpt. at p. 2 describing "The R-11

25

26

27

28   **ORDER RE SUMMARY JUDGMENT-   280**

1 Survey Medical Record Review").[205]

2   Plaintiffs claim that based on the few examples cited by

3 defendants regarding hypothyroidism cases, it is not possible to

4 conclude that Dr. Bird declared as "confirmed," cases which are

5 in fact "unconfirmed." Attached to plaintiffs' Motion to Strike

6 materials are samples of Bird's medical records (Ex. C) which,

7 according to them, show Bird's confirmations are very explicit

8 and that he adequately explained the basis for confirmation in

9 each case.[206]

10   Based on the limited examples and information offered by

11 defendants regarding some of the hypothyroidism cases, the court

12 is not in a position to conclude Bird "routinely" declared

13 reported cases "confirmed" which in fact are unconfirmed. The

14 court is not in a position to declare as "unconfirmed" the

15 particular hypothyroidism cases cited by defendants.[207]

16 _____

17 [205] This section of the report also explains how Bird was
    kept unaware of the status of each patient regarding whether or
    not he/she had been exposed to radiation.

18
    [206] Each of the confirmations provided pertains to **thyroid
19 cancer**, not **hypothyroidism.**

20 [207] In their Motion to Strike materials, plaintiffs do not
    respond to the specific examples regarding hypothyroidism cited
21 by defendants. At the same time, the court's concern is whether
    it has a complete enough picture of the confirmation process from
22 which to make an informed judgment about whether or not the
    medical records are confirmatory. The court does not know what
23 Bird's rationale may have been for confirming these particular
    hypothyroidism cases. Perhaps it is telling that in their Motion
24 to Strike, the plaintiffs cited **thyroid cancer** confirmations
    instead of responding to the specific hypothyroidism examples
25 cited by defendants.
        An additional concern is that in this area (medical
26 confirmation), there would seem to be a need for the assistance
    of some independent medical expertise. Defendants apparently did
27 not retain their own medical expert to offer an opinion about

28 **ORDER RE SUMMARY JUDGMENT-   281**

1   In their Motion to Strike materials, plaintiffs assert Clapp

2 properly used Bird's "confirmation" adjusted numbers, and at each

3 stage of the analysis, statistically adjusted his (Clapp's)

4 findings to assure accuracy. According to plaintiffs, Clapp

5 first adjusted the raw data from the cohort group to account for

6 any error in the self-reporting by study subjects, and secondly,

7 adjusted for "minor differences" between the R-11 study and the

8 NHIS study so they could be meaningfully compared.

9   Clapp's 1995 report and his deposition speak for themselves

10 in establishing that he used unconfirmed self-reported cases, as

11 well as confirmed cases, in arriving at his prevalence rate for

12 non-neoplastic thyroid disease in the R-11 group. Plaintiffs

13 refer to an adjustment to account for any error in the self-

14 reporting by study subjects, but the court fails to see where

15 Clapp describes such an adjustment in his report. Plaintiffs do

16 not specifically cite where this "adjustment" is to be found.[208]

17 Indeed, at his deposition, Clapp testified the only adjustments

18 made with respect to the R-11 Survey and the national survey were

19 for age. (Clapp Dep. at p. 151).

20 _____

21 Bird's confirmations.

22   [208] Plaintiffs claim medical record confirmation of reported
  diseases cures any doubt about Clapp's methodology and his
23 prevalence figures for non-neoplastic thyroid disease. They say
  the extent to which there is in fact such confirmation is
24 relevant to an assessment of the accuracy of those figures and
  also **for determining whether concern about bias in the study**
25 **should in fact be lessened as claimed by Dr. Clapp.**
    Plaintiffs note the NHIS Survey, unlike the R-11 Survey, is
26 based entirely on self-reported cases without any medical
  confirmation. Nonetheless, that is irrelevant because the
27 methodological soundness of the NHIS Survey is not at issue here.

28 **ORDER RE SUMMARY JUDGMENT-   282**

1   Plaintiffs claim defendants' reply brief argument concerning

2 "confirmation adjusted numbers" is a new one and should be

3 stricken. However, in their response brief, plaintiffs

4 specifically asserted that Clapp "adjusted the raw data from the

5 cohort group to account for any error in the self-reporting by

6 the Study subjects." (Plaintiffs' Response Br. at p. 8).

7 Therefore, defendants' reply brief is responsive on this

8 particular point.

9   Finally, defendants note Clapp admitted that even if Bird's

10 confirmation adjustment had been used, it would not cure all the

11 flaws in the R-11 Study. They cite deposition testimony from

12 Clapp in which he stated his concerns about bias "may remain at

13 some level" although "it is certainly diminished by the fact of

14 confirming medical records on a large number of the cases."

15 (Clapp Dep. at p. 259).[209]

16

17   **(6) Daubert Criteria**

18   Considering the methodological flaws of the R-11 Survey **as a**

19 **whole**, the court is overwhelmingly compelled to conclude that the

20 survey is scientifically unreliable.

21   Simply put, the survey was an ill-conceived project from

22 beginning to end: 1) there is no compelling proof that a

23 _____

24   [209] In his November 1997 affidavit (Ex. D to Motion to
   Strike materials), Dr. Javitz claims that if there were
25 substantial biases in the responses of the R-11 Survey
   participants, he would have expected a confirmation rate
26 substantially less than the rate found in the SRI Study (58.1%
   for thyroid problems; 55.7% for malignant neoplasms). See note
27 208 supra.

28
   ORDER RE SUMMARY JUDGMENT-   283

1   qualified epidemiologist was involved in the design of the survey

2   (including selection criteria); it certainly was not Clapp and

3   although the name of Dr. Cummins (Cummings) is thrown about,

4   there is nothing in the record about or from this man; 2) there

5   was no design protocol in place prior to commencement of the

6   survey; 3) correspondence sent to survey participants prior to

7   and during the interview process raises a significant potential

8   the survey results are biased; 4) the absence of a structured

9   questionnaire also raises a significant potential the survey

10   results are biased; 5) plaintiffs have not offered compelling

11   reasons for dropping the control towns, only a conclusory

12   assertion of exposure in those towns; Clapp merely rubber-stamped

13   that decision; 6) there are significant differences between the

14   NHIS Survey and the R-11 Survey, raising questions about their

15   comparability, and also raising questions about when and why the

16   NHIS Survey was chosen (was it just an afterthought?)[210]; 7)

17   medical record confirmation of reported thyroid disease and

18   cancer, to the extent it has occurred and is valid, does not

19   erase the very serious concerns about the reliability of the data

20   collection process and the introduction of bias into the survey.

21   Even Clapp admits there is still doubt: bias "may remain at **some**

22

23   [210]   Note that although Clapp says it was late 1991 or early
1992 when he suggested a "national comparison" as an alternative,
he did not specifically say he had the NHIS Survey in mind at

24   that time. If he had the NHIS in mind, one has to wonder why
there was no effort to make the R-11 Survey more compatible with

25   the NHIS Survey, thereby avoiding the subsequent concerns about
comparability. Likewise, if he had the Connecticut Tumor

26   Registry in mind, it seems more attention would have been paid to
gathering accurate gender information in the R-11 Survey to

27   insure comparability.

28

ORDER RE SUMMARY JUDGMENT-   284

1 level;" medical record verification provides **"some** assurance that

2 questions were asked in a way that got consistent responses."

3   All of these shortcomings must be considered with the

4 <u>Daubert</u> criteria in mind. First of all, there is no question the

5 R-11 Survey was generated for litigation purposes. The

6 plaintiffs cannot and do not claim otherwise. According to

7 plaintiffs' counsel, "[i]t would be disingenuous for plaintiffs

8 to claim that the R-11 Study was done as an abstract exercise for

9 the advancement of science." (Plaintiffs' Response Br. at p.

10 18). The R-11 Survey is not research which has been conducted

11 independent of litigation.

12   Secondly, the R-11 Survey and Clapp's analysis thereof have

13 not been peer reviewed. Plaintiffs admit as much. Nonetheless,

14 they suggest the validity of the survey is borne out because its

15 findings have been referenced by the Agency for Toxic Substances

16 and Disease Registry (ATSDR) as one of the bases for its

17 recommendation of medical monitoring in the Hanford environs.

18 Defendants note, however, ATSDR has only reported that

19 "preliminary results" of the R-11 Study have been made available

20 to the Hanford Health Effects Subcommittee, and that "[w]hen the

21 study is completed and peer reviewed, the findings can be

22 reviewed and considered." (ATSDR 1996 Rpt. at pp. 12 and 64).

23 The plaintiffs are unable to refute this point, arguing only that

24 "[b]ecause the study is ongoing, the final procedures will be

25 written up by Dr. Bird and Dr. Clapp at the conclusion of the [R-

26 11 Study] at which time it will be submitted for publication."

27 (Plaintiffs' Reply Memo re Motion to Strike at p. 30).

28 **ORDER RE SUMMARY JUDGMENT-   285**

1   Plaintiffs argue that "[n]o health organization, in fact no

2 individual, except defendants have come forth with any critique

3 of the survey." That is precisely the point: the R-11 Study has

4 not been peer-reviewed, whether because its results are

5 "preliminary" or for some other reason. Unlike health

6 organizations who can wait for the final results before

7 critiquing the R-11 Study, the defendants do not have such

8 luxury.

9   The two principal ways for showing evidence satisfies the

10 reliability prong of Daubert is if it grows out of pre-litigation

11 research or if the research has been subjected to peer review.

12 Daubert II, 43 F.3d at 1318. The R-11 Survey satisfies neither

13 of these criterion. Furthermore, plaintiffs have cited no

14 evidence that the manner in which the R-11 Survey was conducted

15 would be generally accepted within the scientific community.

16 They have cited no evidence where surveys have been conducted in

17 a similar manner and found scientifically reliable. Plaintiffs

18 suggest medical record confirmation is a satisfactory method for

19 testing survey results and shows the potential rate of error is

20 acceptable. For reasons specified above, the court does not

21 believe medical record confirmation is enough to overcome the

22 numerous flaws which **preceded** the confirmation process.

23   The methodology behind the R-11 Survey is scientifically

24 unreliable. As such, Dr. Clapp cannot reasonably rely upon the

25 survey results in support of his opinions/conclusions.

26 Furthermore, Clapp's use of the NHIS Survey for comparison

27 purposes is methodologically suspect and serves as an additional

28

ORDER RE SUMMARY JUDGMENT-   286

1 (and independent) basis for striking his opinions/conclusions.

2 By the time Clapp really got involved in the R-11 Survey, it was

3 already too late. He could not rehabilitate the survey results.

4

5   **d.   Conclusion**

6   The court will grant defendants' motion in limine regarding

7 the R-11 Survey and the opinions/conclusions of Dr. Clapp based

8 thereon. Granting the motion is justified based on both the

9 "relevancy" and "reliability" prongs of <u>Daubert</u>.

10   The court will deny the plaintiffs' motion to strike

11 portions of the defendants' reply brief. The vast majority of

12 defendants' reply brief is directly responsive to arguments

13 raised in plaintiffs' response brief and does not raise new

14 arguments. Defendants' argument concerning Dr. Bird's summaries

15 (whether they are in fact confirmatory of reported diseases) is

16 arguably new. However, disposition of the motion in limine does

17 not turn on that issue.[211]

18 ───────────────────
       [211]   The non-iodine response brief submitted by <u>Evenson</u>
19 counsel rehashes the unpersuasive arguments offered in their
   response to defendants' motion in limine regarding Dr. Clapp and
20 the R-11 Survey. This <u>Evenson</u> non-iodine response brief was
   filed on November 17, 1997, **after** the defendants filed their
21 reply on the Clapp/R-11 motion in limine (Ct. Rec. 1038 filed
   September 14, 1997). Accordingly, the <u>Evenson</u> non-iodine
22 response doubles as a "surreply" on the motion in limine and
   there is even less reason to grant the <u>Evenson</u> motion to strike
23 defendants' reply on the motion in limine.
       In their non-iodine reply brief, defendants reiterate
24 arguments previously made by them as to the deficiencies of the
   R-11 Survey. However, they make one additional argument not
25 previously offered by them in any of their briefing. Defendants
   assert that whereas the Connecticut Tumor Registry data excludes
26 persons who died before 1982 (the year of the Connecticut study),
   the R-11 Survey includes people who were alive at the time of the
27 survey **and** people who died before the survey. Consequently,
28
   ORDER RE SUMMARY JUDGMENT-   287

1 Plaintiffs complain the Howe affidavit is inappropriate.

2 The court believes it is responsive and an appropriate part of

3 defendants' reply, particularly since the plaintiffs hired an

4 additional expert (Javitz) for their response. In addition, the

5 fact is that Howe merely confirms flaws admitted by Clapp.

6 In their opening brief, defendants asserted that **thyroid**

7 **cancer** prevalence was greater in the control towns than in the

8 study towns. Plaintiffs did not refute that in their response

9 brief. Howe's analysis of thyroid cancer prevalence "clarifies

10 and re-emphasizes" an issue raised in defendants' opening brief.

11 United States v. Birtle, 792 F.2d 846, 848 (9th Cir. 1986). As

12 noted above, plaintiffs' reply memorandum in support of their

13 Motion to Strike is essentially a surreply. Even in that

14 document, plaintiffs do not refute defendants' contention that

15 **thyroid cancer** prevalence was greater in the control towns.

16

17 **7. Compensability of Subclinical Conditions**

18 The defendants contend plaintiffs' claims based on

19 biochemical hypothyroidism, thyroid nodules, and thyroid adenomas

20 are subclinical and/or benign conditions which are not legally

21 compensable "injuries." Defendants assert plaintiffs have no

22 legally cognizable claims for these conditions until they

23 manifest themselves clinically through clinical hypothyroidism or

24 thyroid cancer, or cause some tangible physical impairment.

25 ───────────────────────────
   defendants say the R-11 Survey would overstate thyroid cancer
26 prevalence in comparison to the Connecticut study. (Defendants'
   Non-Iodine Reply Brief at p. 56). The court will not consider
27 this new argument.
28
   ORDER RE SUMMARY JUDGMENT-   288

1 Defendants cite a number of cases, the majority of which deal

2 with subclinical asbestos-related conditions.   None of the cases

3 deal specifically with biochemical hypothyroidism or thyroid

4 nodules and adenomas.

5   Plaintiffs argue that these conditions involve functional

6 impairment, progress to more severe forms, and often require

7 lifetime treatment, and therefore are compensable.   They say the

8 issues raised by defendants are properly adjudicated in the

9 individual damages phase of the proceedings.

10   Washington tort law applies in this case.   Washington courts

11 recognize that plaintiffs cannot recover for **physical** injury

12 unless they are in fact presently suffering from **physical** injury.

13 If they are suffering from present physical injury, they can

14 recover for anxiety as a component of the compensable damages for

15 the physical injury.[212]   Sorenson v. Raymark Industries, 51 Wn.

16 App. 954, 958, 756 P.2d 740 (1988) ("**[Washington] courts have**

17 **long recognized that a plaintiff may recover for anxiety, arising**

18 **from a current reasonable fear of future injury or illness, and**

19 **resulting from an injury caused by the defendant**").   In Sorenson,

20 the plaintiff was diagnosed with asbestosis.   Therefore, because

21 he had suffered an injury resulting from his exposure to

22 asbestos, he was entitled to recover, as an element of his

23 damages, for his reasonable fear of contracting cancer.   Id.

24   To recover for **physical injury**, there must be a present

25

_____

26   [212] As distinguished from recovery only for present
emotional harm sought via a claim for intentional or negligent
27 infliction of emotional distress.

28
ORDER RE SUMMARY JUDGMENT-   289

1 manifestation of **physical injury**. The law, however, cannot

2 answer the question of whether a condition constitutes present

3 physical injury. That can only be answered by science and

4 medicine. Unfortunately, no cases have specifically analyzed the

5 scientific and medical evidence and reached a conclusion about

6 the compensability of biochemical hypothyroidism and benign

7 thyroid nodules and adenomas as **physical injuries**.

8   Defendants assert plaintiffs' own experts opine these

9 conditions do not amount to physical injury. Dr. Ruttenber was

10 asked the medical significance of subclinical hypothyroidism and

11 responded that "it could develop into hypothyroidism." When

12 asked whether it was a condition that should be watched,

13 Ruttenber stated: "Well, you know, that's depending on who you

14 talk to, and I'm not a clinician, and I just really don't want to

15 speak to that . . . . I've heard other people say . . . watch it

16 symptomatically. I don't know." (Ruttenber Depo. at pp. 120-

17 21). When asked the clinical significance of "a single [thyroid]

18 nodule," Ruttenber responded:

19   . . . the clinical significance of a thyroid
            adenoma or a benign neoplasm of the thyroid is
20   it may turn into a carcinoma. If it's just a
            benign nodule, there may not be any clinical
21   significance, but it has more significance in
            terms of being an indicator of a neoplastic
22   process.

23 (Ruttenber Depo. at pp. 174-75).

24   Ruttenber was very careful to qualify his remarks about the

25 "clinical significance" of biochemical hypothyroidism and benign

26 thyroid nodules and adenomas. He emphasized he was not a

27 "clinician" and could only speculate about the "clinical

28
ORDER RE SUMMARY JUDGMENT-   290

1 significance" of these conditions. In his 1995 iodine report,

2 Ruttenber did not discuss whether these conditions amount to

3 present functional impairments. Therefore, the court is not

4 convinced he has concluded, as defendants claim, that there is no

5 "clinical significance" to these conditions.

6   The plaintiffs offer a declaration from Michael Lawson, M.D.

7 dated July 7, 1997. (Exhibit 4 to Appendix 1 re Iodine Claims).

8 According to Lawson, the plaintiffs retained him to review "the

9 issues which the defendants have raised regarding thyroid

10 nodules, thyroid adenomas and subclinical hypothyroidism." The

11 defendants move to strike the Lawson declaration on the basis

12 that he never submitted an expert report in this case and

13 therefore, his declaration is not properly a part of the summary

14 judgment proceedings.

15   Lawson indicates medical treatment is appropriate for benign

16 thyroid nodules and adenomas. This is because there is a "risk

17 of malignancy in individual nodules whether they occur singularly

18 or in the context of a multinodular gland or adenomatous goiter."

19 According to Lawson:

20   A diagnosis of malignancy can be established
                with a fine needle aspiration biopsy (FNA) or
21   with excision of a suspect nodule followed
                by a cytological examination. Malignancy, however,
22   is difficult to disprove even in the context of a
                negative biopsy. Experts often recommend a
23   repeat FNA at interval of one year. Follow-up
                evaluations are also recommended by a majority
24   of thyroid experts who will usually refer a
                patient for surgical excision when a nodule is
25   enlarging even if a negative FNA has previously
                been obtained.
26
      A second concern of nodules and adenomas, says Lawson, is
27
28
   ORDER RE SUMMARY JUDGMENT-   291

1 that of thyroid dysfunction which can manifest itself as either

2 thyroid hyperfunction or hypofunction. Because these conditions

3 can develop over time and cannot be excluded by any single

4 evaluation, he says a specific "follow-up" program is necessary.

5   In his July 4, 1997 declaration, Dr. Radford indicates that

6 nodules and adenomas which are large enough can cause pain and

7 prevent proper fitting of neckwear. (Ex. 7 to Appendix 1 re

8 Iodine Claims at Paragraph 5).

9   With regard to subclinical hypothyroidism, Lawson states

10 this condition is characterized by an elevated level of

11 thyrotropin (TSH) which cannot otherwise be explained. He says

12 this condition often occurs in the presence of elevated

13 antithyroid antibodies which are markers of an underlying

14 autoimmune process. Lawson opines that patients with subclinical

15 hypothyroidism warrant continued follow-up and treatment with

16 thyroid hormone in the form of L-thyroxine (T4) to normalize

17 their TSH level. (Ex. 4 to Appendix 1 at Paragraph 5).

18   In their response brief, plaintiffs cite a number of medical

19 journal articles which discuss symptoms of subclinical

20 hypothyroidism.[213] Defendants contend these articles are not

21 mentioned in any of plaintiffs' expert reports. Some of the

22 ───────────────

23 [213] The articles include: Arem, R. and Escalante, D.,
    "Subclinical hypothyroidism: epidemiology, diagnosis and
    significance," 41 **Adv Intern Med** 213-50 (1996); Tibaldi, J. and

24 Barzel, U.S., "Thyroxine supplementation: method of the
    prevention of clinical hypothyroidism," 79 **Am J Med** 241-44

25 (1985); Kabadi UM, "Subclinical hypothyroidism: Natural course
    of the syndrome during a prolonged follow-up study," 153 **Arch**

26 **Intern Med** 957-61 (1993); Rosenthal MJ, et al. "Thyroid failure
    in the elderly- microsomal antibodies as discriminant for

27 therapy," 258 **JAMA** 209-13 (1987).

28 **ORDER RE SUMMARY JUDGMENT-**   **292**

1 symptoms discussed in the articles include easy fatigability, dry

2 skin, hair loss, weight gain, cold intolerance, cognitive and

3 memory deficits, depression, infertility, spontaneous abortions,

4 etc.   These articles also report that subclinical hypothyroidism

5 can lead to more serious conditions including atherosclerotic

6 cardiovascular disease and, of course, clinical hypothyroidism.

7   Even if the court did not consider the Lawson and Radford

8 declarations, or the journal articles, it could not justify

9 finding as a matter of law that subclinical hypothyroidism and

10 thyroid nodules and adenomas can never constitute compensable

11 **physical** injury.   Defendants, as the moving party, have not met

12 their initial burden of proving the absence of a genuine issue of

13 material fact.   Dr. Ruttenber's testimony and the caselaw cited

14 by defendants is insufficient for that purpose.

15   A claim for subclinical hypothyroidism or thyroid nodules

16 and adenomas cannot be heard by a jury unless there is sufficient

17 evidence from which an inference is raised that radioiodine

18 exposure is a "more likely than not" cause of those conditions.

19 Consequently, a jury may not consider whether an individual's

20 subclinical hypothyroidism or thyroid nodules and adenomas should

21 be compensated as **physical injuries, unless** the individual can

22 **first** prove exposure to a dose of radioiodine which more than

23 doubles his risk of developing those conditions.   In this case,

24 the doubling doses for thyroid cancer will apply to claims

25 involving thyroid nodules and adenomas.   Dr. Ruttenber has

26 provided a doubling dose specifically for subclinical

27 hypothyroidism.

28

ORDER RE SUMMARY JUDGMENT-   293

1   Furthermore, even if subclinical hypothyroidism and benign

2 thyroid nodules and adenomas are not compensable as physical

3 injuries, they are potentially compensable under an emotional

4 distress theory if an individual has a **reasonable** fear of

5 developing clinical hypothyroidism or thyroid cancer because of

6 exposure to Hanford emissions. Sorenson v. Raymark Industries,

7 cited supra.[214] Plaintiffs' complaint includes claims for

8 intentional and negligent infliction of emotional distress,

9 neither of which depend on physical injury. Rice v. Janovich,

10 109 Wn. 2d 48, 61, 742 P.2d 1230 (1987); Hunsley v. Giard, 87 Wn.

11 2d 424, 435 (1976). Recovery for emotional distress requires

12 only some objective symptom of such distress and the victim's

13 reaction must be reasonable under the circumstances.

14   Whether an individual has a reasonable fear of developing

15 thyroid cancer or hypothyroidism because of exposure to Hanford

16 emissions obviously depends upon the extent of his exposure. An

17 emotional distress claim cannot be heard by a jury unless there

18 is sufficient evidence from which an inference can be raised that

19 the fear is reasonable.

20   For example, an individual who fears developing thyroid

21 cancer cannot recover unless there is evidence he has been

22 exposed to a dose of radioiodine in excess of the doubling dose

23 for thyroid cancer. Fear of developing thyroid cancer **because of**

24 **exposure to Hanford emissions** is not reasonable unless the risk

25 of developing thyroid cancer is more than doubled because of that

26 _____

[214] The defendants concede these conditions are the proper
27 subject of plaintiffs' medical monitoring claims.

28 **ORDER RE SUMMARY JUDGMENT-   294**

1 exposure. An individual with thyroid nodules and adenomas (or

2 for that matter, an individual without nodules and adenomas) who

3 has been exposed to a dose of Hanford emissions in excess of the

4 doubling dose may have a reasonable fear of developing thyroid

5 cancer because of that exposure.

6

7   **8. Summary**

8   Based on plaintiffs' expert evidence, the only radioiodine

9 claims which can potentially be heard by a jury are those for

10 thyroid cancer and non-autoimmune hypothyroidism. Thyroid cancer

11 claims (including claims for thyroid nodules and adenomas) cannot

12 survive summary judgment unless there is proof of exposure in

13 excess of the following doubling doses derived from Dr. Radford's

14 work: 5 rads for those 0 to 4 at the time of exposure; 10 rads

15 for those 5 to 9 at the time of exposure; 33 rads for those 10 to

16 19 at the time of exposure; and 100 rads for those 20 and over at

17 the time of exposure.

18   Clinical (non-autoimmune) hypothyroidism claims cannot

19 survive summary judgment unless there is proof of exposure in

20 excess of 750 rads, the doubling dose opined by Dr. Ruttenber.

21 Subclinical (non-autoimmune) hypothyroidism claims cannot survive

22 summary judgment unless there is proof of exposure in excess of

23 350 rads.[215]

24 _____

25   [215] A DREF is not incorporated in these figures for the
   reasons discussed <u>supra</u> in conjunction with Dr. Radford's opinion
   that there is equal effectiveness between external and internally
26 deposited radiation.
       Ruttenber states his clinical and subclinical hypothyroidism
27 doubling doses are for the **non-autoimmune** variety of these

28

ORDER RE SUMMARY JUDGMENT-   295

1   Presumably because Hanford radioiodine emissions

2 significantly diminished after 1960, plaintiffs' counsel concede

3 they are not pursuing claims for individuals whose radioiodine

4 exposure occurred **entirely** after 1960. Accordingly, at least a

5 portion of an individual's radioiodine exposure must have

6 occurred prior to the expiration of 1960.[216]

7

8   **B.   Columbia River Emissions**

9   **1.   Hexavalent Chromium**

10   **a.   Introduction**

11   Plaintiffs' assert claims for gastrointestinal (GI) cancer

12 based on exposure to hexavalent chromium (aka "Cr VI").

13 Hanford's original eight reactors were cooled by filtered,

14 chemically-treated water from the Columbia River which was held

15 for a period of time in retention basins, then returned to the

16 Columbia River. To inhibit corrosion of reactor piping during

17 the cooling process, Hanford's operators sometimes added sodium

18 dichromate to the river water before it entered the reactor.

19 Sodium dichromate contains hexavalent chromium. Consequently,

20 when the cooling water was returned to the river, it contained

21 hexavalent chromium. Hexavalent chromium is not a radioactive

22 substance.

23   Plaintiffs' hexavalent chromium case is premised on two

24 expert reports, one by Dale Hattis, Ph.D, and one by Sidney A.

25 _____

conditions.

26

[216] This includes in utero exposures prior to the expiration

27 of 1960.

28
ORDER RE SUMMARY JUDGMENT-   296

1 Katz, Ph.D. Dr. Hattis is a geneticist. He is currently a

2 Research Associate Professor with the Center for Technology,

3 Environment and Development in the George Perkins Marsh Institute

4 at Clark University. He specializes in the analysis of

5 variability and uncertainty in the context of environmental and

6 occupational health risk assessments. Dr. Katz is a chemist. He

7 is a professor of chemistry at Rutgers University. The

8 defendants do not take issue with the qualification of either

9 Hattis or Katz to offer the opinions contained in their

10 respective reports.

11

12   **(1) Katz Report**

13   Katz submitted a report dated April 1, 1996 which is

14 entitled "Chromium Toxicology." It contains an overview of

15 chromium chemistry. Katz describes the various chromium

16 compounds, including hexavalent chromium (Cr6) which he states

17 has "higher potentials for harming human health and environmental

18 quality than do compounds of trivalent chromium [Cr3]." (Katz

19 Rpt. at p. 1). Katz describes the toxicity of hexavalent

20 chromium versus trivalent chromium in a number of different

21 contexts: acute oral toxicity, dermal toxicity, cryotoxicity,

22 genotoxicity and carcinogenicity. According to Katz, "in

23 general, the hexavalent forms are more toxic than the trivalent

24 forms."

25   Katz notes that bronchiogenic cancer "appears to be

26 associated" with the **inhalation** of hexavalent chromium compounds,

27 and that on the basis of tumor incidence in the chromate-

28 **ORDER RE SUMMARY JUDGMENT-   297**

1 producing industry, "cancer of the lung in humans has been

2 attributed to the **inhalation** of hexavalent chromium compounds."

3 (Id. at pp. 4-5). He notes that **"non-cancer** systemic effects of

4 hexavalent chromium include toxicity to the kidney, liver and

5 lung," and that hexavalent chromium compounds have been "reported

6 to damage the developing fetus in animals." (Id. at p. 5).[217]

7   Katz concludes his report with a section specifically

8 devoted to "Chromium Hazards at Hanford." He discusses the

9 amount of hexavalent chromium discharged into the river and

10 asserts that "non-radioactive chromium [hexavalent chromium] from

11 the coolant exhausted into the Columbia River represents a long-

12 term hazard to public health and environmental quality." He adds

13 that mobilization of chromium from groundwater sources located

14 below the retention basins, and chromium already deposited in

15 river sediments, also "represent[] a long-term hazard to public

16 health and environmental quality." (Id. at pp. 6-7).

17   Katz was asked by plaintiffs' counsel to comment on possible

18 "synergistic" effects of chromium with other substances

19 discharged into the river, including radioactive plutonium. In

20 an April 4, 1996 letter addressed to plaintiffs' counsel, Katz

21 _____

22   [217] In their response brief, plaintiffs suggest some of
their claims are based on non-carcinogenic chromium poisoning as

23 opposed to GI cancer. They also allude to there being some birth
defect claims based on exposure to hexavalent chromium. However,

24 Dr. Hattis limits his health effects analysis to GI cancer.
     In any event, claims based on chromium poisoning and birth

25 defects would have to be dismissed because there is no assessment
of risk. Furthermore, with regard to birth defects, the

26 plaintiffs have not presented sufficient evidence to raise a
genuine issue of material fact that hexavalent chromium is even

27 "capable of causing" birth defects in **humans.**

28 **ORDER RE SUMMARY JUDGMENT-   298**

1 stated he found no reports on multifactorial cancer risks for

2 exposure to chromium and plutonium, or for exposure to chromium

3 and arsenic. He was also unable to find any reports on studies

4 connecting chromium exposure and **radiogenic** carcinoma.[218]

5 Nonetheless, Katz ventured to say counsel was "probably correct"

6 that "radiation insult coupled with chemical insult could well

7 increase the cancer risk."

8

9 **(2) Hattis Report**

10 In his March 29, 1996 report[219], Dr. Hattis analyzed the

11 population risk associated with **ingestion** of hexavalent

12 chromium[220] by first defining his assumed exposed population;

13 secondly, estimating population dose; and thirdly, selecting a

14 risk co-efficient (risk of GI cancer per unit of dose). He then

15 multiplied his risk co-efficient by the population dose to

16 generate an estimated number of cancers.

17 Hattis' assumed exposed population consists of four cities:

18 Boardman, Oregon; Richland, Washington; Kennewick, Washington and

19 Pasco, Washington. According to Hattis, his "current"

20 information was that these were the only cities known to have

21 _____

22 [218] Presumably this refers to chromium exposure in
combination with radiation exposure. Cancers can be radiogenic
23 (radiation-induced) or non-radiogenic.

24 [219] The report is entitled "Assessment of Population
Aggregate Cancer Risks From Radionuclides and Chromate Released
25 by Hanford Operations into the Columbia River."

26 [220] Ingestion by way of drinking river water or eating fish
caught from the river. Inhalation of hexavalent chromium is not
27 an issue.

28

ORDER RE SUMMARY JUDGMENT-   299

1 used the Columbia River as the direct source of their drinking

2 water. Hattis reported the average concentration of hexavalent

3 chromium in the Columbia River was 4.69 micrograms per liter in

4 Boardman from January 1950 to January 1971; 5.66 micrograms per

5 liter in Richland from October 1963 to January 1971; and 6.97

6 micrograms per liter in Pasco and Kennewick from January 1950 to

7 January 1971. (Hattis Rpt. 1996 at p. 27). Defendants, for

8 purpose of this motion, do not challenge these concentration

9 estimates.

10   Hattis concludes that between January 1950 and January 31,

11 1971, the aggregate dose of hexavalent chromium received by all

12 61,940 persons[221] living in the four study communities was "1.56

13 People*lifetime mg/kg-day." The aggregate population dose

14 represents the sum of the dose estimates for each of the four

15 communities. Hattis converted the concentration estimates for

16 each community into average "lifetime equivalent dose[s]" per

17 kilogram of body weight. The doses assume that each day of the

18 relevant time period the average person in the four communities

19 drank 1.2 liters of river water and weighed 70 kilograms. The

20 average doses for each community were multiplied by the

21 population of that community to obtain a community population

22 dose. (Table 11 of Hattis 1996 Rpt. at p. 27).

23   Hattis then multiplied his aggregate population dose (1.56

24 People*lifetime mg/kg-day) by three risk-coefficients (0.125;

25

---

26 [221] 25,600 persons in Richland; 34,900 persons in
Pasco/Kennewick; and 1,440 persons in Boardman. This equals 61,
27 940 persons.
28
ORDER RE SUMMARY JUDGMENT-   300

0.42; and 13) to generate an estimated number of GI cancers potentially attributable to hexavalent chromium. These risk co-efficients were derived from animal data, specifically a mouse study. For two of the risk co-efficients (0.125 and 0.42) the projected number of GI cancers was less than one (0.2 and 0.65) respectively. The third risk co-efficient resulted in a projected excess of 21 cancers. (Table 12 of Hattis 1996 Rpt. at p. 30). Hattis concluded his report by stating that Table 12 provides "a plausible range of cancer cases from drinking water ingestion of hexavalent chromium rang[ing] from less than 1 to over 10 cases." (Id. at p. 32).[222]

b. Daubert Analysis

Defendants seek summary judgment on all plaintiffs' health claims which are based on alleged exposure to hexavalent chromium. Defendants assert summary judgment is justified because the expert opinions of Hattis and Katz, on which plaintiffs' claims are premised, flunk the Daubert test.

---

[222] In his report, Hattis identified a "potential" alternative approach to assessing risk by using existing epidemiological studies (human data) to compare excess GI cancers with excess lung cancers. However, Hattis cautioned that his preliminary result (Table 13, Hattis 1996 Rpt. at p. 31), suggesting GI cancer risks might be appreciable in relation to lung cancer risks from **inhalation exposures** to hexavalent chromium, "may not be sustained at the same level in a more comprehensive examination of the epidemiological literature." (Id. at p. 32). Furthermore, Hattis indicated this approach merely possessed "some promise to shed light on the likely comparative potency of hexavalent chromium by ingestion and inhalation." (Id. at p. 30).

ORDER RE SUMMARY JUDGMENT-   301

(1) Fit/Relevancy

Defendants claim Hattis' proposed testimony is irrelevant to the plaintiffs' causation burden of proof because he does not opine that hexavalent chromium doses from the Columbia River "more than doubled the risk" of GI cancer, or that any person could prove causation at the doses assumed in his analysis.

Defendants note that at his deposition, Hattis described the figures contained in Table 12 of his report as not defining a "best estimate of risk," but a "broad range of not clearly incorrect answers." (Hattis Dep. at p. 204). This range is between less than one cancer and as many as 21 cancers.

Obviously, Hattis is not willing to stake his analysis on there being an excess of 21 GI cancers due to hexavalent chromium exposure. He is just as willing to venture the result might be zero excess cancers, which is the result derived from two of his three risk co-efficients. Zero excess cancers clearly does not constitute a "doubling of the risk." This type of evidence does not allow a jury to infer it is "more likely than not" that anyone's GI cancer was caused by exposure to hexavalent chromium. Rather, it invites a jury to speculate whether it is "more likely than not" so.

Plaintiffs' burden of proof at this stage of the proceedings is to show the doses[223] at which it is "more likely than not"

_____

[223] With hexavalent chromium, doses are not measured in rads, but in a drinking water dose (1.56 person- mg/kg- day population aggregate drinking water dose). Defendants point out that Hattis does not provide a model for determining individual dose or risk, nor a method for proving the doses allegedly at issue caused any plaintiff's injury. Plaintiffs do not dispute

ORDER RE SUMMARY JUDGMENT-   302

1   hexavalent chromium exposure causes GI cancer (or any other

2   health effects). Consequently, the proffered testimony of Dr.

3   Hattis does not "fit" and will be excluded based on Prong 2 of

4   Daubert.

5   The same is true with respect to Dr. Katz. Katz does not

6   address chromium concentrations at the levels alleged to exist in

7   the Columbia River and does not attempt to tie those

8   concentrations to any health effects. Furthermore, in his risk

9   analysis, Hattis places no reliance on Katz. Indeed, Hattis does

10 not even mention Katz' report.

11   Plaintiffs, of course, contend "doubling of the risk" is not

12 the standard at this "generic" causation stage of the

13 proceedings. Plaintiffs assert: "[T]estimony that the chromium

14 compounds that Hanford added to the reactor cooling water posed a

15 longtime health hazard which will cause an **increase[d] risk** of

16 cancers to members of the aggregate downwinder/downriver

17 population is both relevant and admissible in this action."

18 Plaintiffs add that testimony concerning "the pathogenesis and

19 health effects as well as the supporting literature and studies

20 will assist the trier of fact in understanding the various cancer

21 mechanisms affiliated with exposure to hexavalent chromium."

22   As will be discussed, the court is not convinced that

23 Hattis' opinion raises an issue of material fact that **ingestion**

24 of hexavalent chromium is even "capable of causing" GI cancer

25

26 _____

    that point, but argue those considerations are appropriately left
27 for the individual causation stage of the proceedings.

28 **ORDER RE SUMMARY JUDGMENT-   303**

such that there is some increase in the risk of such cancer.[224]

In his report, Dr. Katz discusses bronchiogenic cancer which he says "appears to be associated" with the **inhalation** of hexavalent chromium compounds, and lung cancer which "has been attributed" to **inhalation** of hexavalent chromium compounds. "Inhalation" of hexavalent chromium is not an issue in this case. The issue is "ingestion" through consumption of drinking water and the eating of fish from the Columbia River.

Katz discusses **"non-cancer** systemic effects of hexavalent chromium" including toxicity to the kidney, liver and lung. This appears to based on ingestion of Cr VI since Katz refers in general to animal studies showing the "chronic **oral RfD"** (reference dose) for hexavalent chromium is 0.005 mg/kg/day. (Katz Rpt. at p. 5). Nevertheless, the fact is neither Katz or Hattis offer any type of risk assessment purporting to show the extent to which hexavalent chromium ingestion is the "more likely than not" cause of any **"non-cancer** systemic effects."[225]

Katz offers a solitary sentence that hexavalent chromium compounds have been "reported to damage the developing fetus in animals." (Katz Rpt. at p. 5).[226] He never says anything about a causal connection between hexavalent chromium exposure and

---

[224] Obviously, an increase in risk does not necessarily amount to a doubling of the risk.

[225] Defendants do not specifically dispute that hexavalent chromium (via ingestion or inhalation) is "capable of causing" toxicity to the kidney, liver or lung.

[226] He likewise offers a one sentence analysis that hexavalent chromium compounds "include mutagenic effects." (Katz Rpt. at p. 5).

ORDER RE SUMMARY JUDGMENT—   304

birth defects in **humans**. Katz' report clearly does not

constitute a scientifically reliable analysis that ingested

hexavalent chromium is "capable of causing" birth defects in

humans.[227] Besides that, neither Katz or Hattis make any

assessment of the risk that hexavalent chromium exposure (via

ingestion) is the cause of human birth defects.

As noted above, Katz stated he found no reports on

multifactorial cancer risks for exposure to chromium and

plutonium, or for exposure to chromium and arsenic. He was also

unable to find any reports on studies connecting chromium

exposure and **radiogenic** carcinoma. Nonetheless, Katz was willing

to say counsel was "probably correct" that "radiation insult

coupled with chemical insult could well increase the cancer

risk." This nowhere approaches a scientifically reliable

analysis that there is a "synergistic" effect between chromium

and radiation (or anything else). It amounts to no evidence

whatsoever that hexavalent chromium exposure in combination with

radiation exposure increases the risk of any health effect

compared to radiation exposure alone.

Katz' report is wholly irrelevant, not only for the

proposition that ingested hexavalent chromium is the "more likely

than not" cause of any health effect, but also for the

_____

[227] Even plaintiffs recognize the speculative nature of
Katz' report as support for a causal connection between
hexavalent chromium exposure and human birth defects. In their
brief, plaintiffs assert Katz' testimony will "assist the trier
of fact as to the **possible** etiology of birth defects as related
to chromium exposure." (Plaintiffs' Response Br. at p.
5)(Emphasis added).

ORDER RE SUMMARY JUDGMENT-   305

1   proposition that it is "capable of causing" any particular health

2   effect in humans.   Having Dr. Katz testify in general about the

3   biological and environmental chemistry of chromium is of no

4   assistance whatsoever to a jury in determining whether hexavalent

5   chromium is "capable of causing" certain health effects, or more

6   importantly, whether it is a "more likely than not" cause of

7   certain health effects.

8

9   **(2)   Reliability**

10   **(a)   Borneff Mouse Study**

11   It is undisputed that no epidemiological study or animal

12 study has found ingested hexavalent chromium is carcinogenic.   In

13 his report, Hattis acknowledges that although hexavalent chromium

14 "is well established as a human carcinogen by the **inhalation**

15 route . . . its activity by the **oral** route is more uncertain and

16 controversial."   (Hattis 1996 Rpt. at p. 4)(Emphasis added).

17 According to Hattis:

18   [T]here is good reason to expect that
            hexavalent chromium is considerably less
19   [potent] by ingestion than it is by in-
            halation.   In the stomach, ingested
20   chromium encounters an acidic environ-
            ment and other factors that reduce it
21   to trivalent chromium at an appreciable
            rate.   Although it is an error to infer
22   that this kind of process can produce a
            true threshold in the dose response relation-
23   ship, the fraction effectively absorbed
            by cells would be expected to be less than
24   if the same amount of hexavalent chromium
            were deposited in the lung.   How much less
25   has evidently not yet been the subject of
            extensive measurement and/or modeling in
26   people, although a promising pharmacokinetic
            model had been developed for rats.   With
27   further study, it might be possible to

28 **ORDER RE SUMMARY JUDGMENT-   306**

1   adapt this model to predict the comparative
         pharmacokinetics of hexavalent chromium in
2   people.

3 (Hattis 1996 Rpt. at pp. 27-28).   Clearly, Hattis can only

4 speculate about the comparative potency of inhalation and

5 ingestion.

6   It is against this background that Hattis comes to rely, at

7 least indirectly, on the Borneff mouse study[228] as the basis for

8 his risk co-efficients.   Hattis describes the study as "an older

9 and less-than-ideal lifetime study of drinking water exposure to

10 hexavalent chromium compound in mice."   At his deposition, Hattis

11 admitted he had not actually read the Borneff mouse study itself,

12 but only a summary prepared by the California Environmental

13 Protection Agency.   (Hattis Dep. at p. 199).   In his report,

14 Hattis describes how the California EPA, using the Borneff study,

15 calculated an upper confidence limit risk for the ingestion route

16 of 0.42 (mg/kg-day lifetime exposure).   According to Hattis, this

17 figure is 1000 times less than the corresponding value for

18 **inhalation** potency derived from the human epidemiology.   (Hattis

19 Rpt. at p. 28).

20   The Borneff study involved three generations of mice exposed

21 to potassium chromate in drinking water at a concentration of 500

22 mg/L for 880 days.[229]   Potassium chromate contains hexavalent

23 _____

24   [228] Borneff, J. et al., "Kanzerogene Substanzen in Wasser und
   Boden" [Carcinogenic Substances in Water and Soil], 152 **Arch Hyg**
   45 (1968).   Defendants' Ex. 154.

25

26   [229]   The defendants hired their own expert, Joseph Rodricks,
   Ph.D., a biochemist, to critique the reports of Hattis and Katz.
   In his report, Rodricks describes the Borneff mouse study and the
27 results thereof.   Plaintiffs do not dispute Rodricks' description

28 **ORDER RE SUMMARY JUDGMENT-   307**

chromium. The exposure was reported to be equivalent to a dose

of 1 mg potassium chromate per day (0.26 mg hexavalent

chromium/day). For a mouse with an average body weight of 31

grams, this corresponded to a dose of 8.4 mg hexavalent

chromium/kg/day. Defendants note this dose is substantially

greater than the 1.56 mg/kg-day lifetime exposure aggregate

population dose used by Hattis in predicting excess cancers in

humans.

Approximately two-thirds of the mice died between the eighth

and eleventh month of the study due to a "mouse pox" epidemic.

The researchers were left with 66 females and 35 males from the

dosed (exposed) group, and 79 females and 47 males from the

control (unexposed) group. The mice were killed and necropsies

were performed to determine tumor incidence among them. The

results were as follows: 1) no forestomach cancers in either

group of male mice; 2) two forestomach cancers in the exposed

females and none in the controls; 3) benign forestomach tumors or

non-tumor lesions in one exposed male and in three control males;

and 4) benign forestomach tumors or **non-tumor lesions** in nine

exposed females and two control females. (Rodricks Rpt. at pp.

15-16).

The Borneff study researchers concluded that "[o]rally

administered chromate does not always have a carcinogenic effect

on mice." They added that the existence of two stomach cancers

in 101 animals (the exposed group- 66 females and 35 males),

of the Borneff study.

ORDER RE SUMMARY JUDGMENT-   308

1 provided "an indication that further tests must be carried out."

2 (Borneff, et al., 1968 at p. 12; Defendants' Ex. 154). In other

3 words, the researchers concluded their results were not

4 sufficient support for the proposition that ingested hexavalent

5 chromium causes cancer in mice, let alone humans. The results

6 were inconclusive. The Agency for Toxic Substances and Disease

7 Registry described the results of the Borneff study as follows:

8   No evidence of carcinogenicity was found in
           mice exposed to potassium chromate in drinking
9   water at 9 mg chromium (VI)/kg/day for three
           generations (880 days).
10
(ATSDR 1993 at p. 25; Defendants' Ex. 160).
11

12
       (i)   First Risk Co-efficient
13
       Hattis used the Borneff mouse data to produce his first risk
14
co-efficient- 0.125- which yielded a result of 0.2 (zero) excess
15
cancers (1.56 aggregate population dose x 0.125). He described
16
his calculation approach as: "Simple one-hit maximum likelihood
17
fit to mouse data, body weight 3/4 dose projection to people."
18

19
       (ii)   Second Risk Co-efficient
20
       For his second risk co-efficient, Hattis used the "upper
21
confidence limit risk" calculated by the California EPA for the
22
ingestion route- 0.42.[230] This yielded a result of 0.65 (zero)
23
excess cancers (1.56 x 0.42). As noted above, the California EPA
24
derived its figure from the Borneff mouse study. The 0.42 figure
25
_____

26   [230] Hattis describes the calculation as: "Upper confidence
limit fit to the mouse data, surface area dose projection to
27 people."

28 **ORDER RE SUMMARY JUDGMENT-   309**

comes from a **recommendation** by the "Standards and Criteria
Workgroup," (hereinafter, the "workgroup"), of the California EPA
(formerly part of the Department of Health Services).

In May 1990, the workgroup concluded there was sufficient
scientific information to treat hexavalent chromium as a
carcinogen by the oral route. However, it acknowledged it was
unable to identify a published bioassay using laboratory animals
or epidemiological study that by itself was adequate to establish
the carcinogenicity of hexavalent chromium by the oral route.
Nonetheless, "based on the weight of the evidence and to be
consistent with the responsibility to protect public health," it
recommended hexavalent chromium be "assumed" as a human
carcinogen by the oral route. (Defendants' Ex. 156; August 7,
1990 memo at p. 4).

The conclusion of the workgroup was reiterated in a May 1991
memo in which it came up with the 0.42 figure based on the
Borneff mouse study. The workgroup observed the increased
incidence of malignant stomach tumors was not significant, but
the incidence of malignant **and** benign tumors in the dosed females
(11/66) was significantly increased above the incidence in the
controls (2/79). Thus, the workgroup combined the incidence of
malignant and benign tumors in arriving at their 0.42 figure.
(Defendants' Ex. 155; May 30, 1991 memo at p. 2). The workgroup
once again recognized further research on the carcinogenic
potency of hexavalent chromium was needed. (Id. at p. 4).

Defendants note the figure developed by the workgroup has
not yet been used as the basis for a regulatory standard in

ORDER RE SUMMARY JUDGMENT-   310

1   California.   (Defendants' Ex. 157 at p. 20).   Neither has the

2   figure been peer-reviewed in any scientific journal, according to

3   Hattis.   (Hattis Dep. at p. 207).

4

5   **(iii)   Third Risk Co-efficient**

6   Although defendants suggest there are methodological

7   shortcomings in the derivation of the 0.42 figure, it is Hattis'

8   third risk co-efficient which is the center of controversy.   Even

9   assuming the 0.42 figure is reliable, it generates less than one

10   excess cancer (zero) according to Hattis' analysis.   On the other

11   hand, Hattis' third risk co-efficient (13) yields a total of 21

12   excess cancers (1.56 x 13).[231]

13   Hattis started out with the California EPA **inhalation**

14   potency figure for hexavalent chromium in humans: 510 mg/kg/per

15   day.   To apply this potency estimate to hexavalent chromium by

16   **ingestion**, Hattis developed a "potency ratio" to establish a

17   comparative relationship between human potency by ingestion and

18   human potency by inhalation.   This "potency ratio" was the result

19   of a comparison between a potency estimate of 16 mg/kg-day,

20   derived by the California EPA from a rat inhalation study

21   (Glaser, et al., 1986), with the potency estimate of 0.42 mg/kg-

22   day derived from the Borneff study.   0.42 is divided by 16 to

23   obtain a ratio of 1/40, meaning that for rodents the cancer

24   potency of hexavalent chromium by ingestion is 1/40 the cancer

25   _____

26   [231]   Hattis describes this calculation approach as: "Mouse
    ingestion/rat inhalation upper confidence limit potency ratio,
    multiplied by the California upper confidence limit estimate of
27   inhalation potency for people."

28   **ORDER RE SUMMARY JUDGMENT-   311**

potency by inhalation. According to Hattis, 0.42 is "about 40 times less than the value calculated from the rat inhalation experiment." (Hattis 1996 Rpt. at p. 28). Hattis then multiplied the 1/40 "rodent ratio" by 510 (the human cancer potency reported by California EPA for inhalation of hexavalent chromium) to arrive at an **ingestion potency** of 13. Hattis "assumes" the 1/40 rodent ingestion to rodent inhalation potency ratio is comparable to the ratio of ingestion to inhalation potency in humans. (Hattis Rpt. at pp. 28-29; Hattis Dep. at pp. 202-04).

Defendants assert Hattis' risk co-efficient of 13 is not scientifically reliable because it is not supported by **any** epidemiological study or animal study establishing that ingested hexavalent chromium causes GI cancer. Defendants note that Hattis extrapolates from the Borneff mouse study which did not find hexavalent chromium ingestion causes GI cancer.

Indeed, Hattis' deposition testimony indicates because of his reliance on the summary of the Borneff study prepared by the California EPA, he was not even aware the Borneff researchers had not found a causal connection between hexavalent chromium ingestion and GI cancer. (Hattis Dep. at pp. 199-200). Hattis downplayed the importance of the Borneff study conclusion:

> The opinions of the authors of a particular study have some weight but not a definitive weight in the analyses that I do. I often use people's data for purposes they didn't envision. [I]t's based in part upon my qualitative under-standing of mechanisms of cancer and my judgment that if we have a local site acting carcinogen that's probably acting by a DNA involved mechanism, that some amount of chromium VI will probably

ORDER RE SUMMARY JUDGMENT-   312

1   get into epithelial cells of the lining of the
2   stomach and it will have some carcinogenic
             activity very likely. How much is a very open
3   question and a very controversial question as I
             think I pointed out in the document but it
4   does not depend solely on the Borneff study.

(Hattis Dep. at pp. 201-02).

Hattis apparently refers to what he considers the

"biological plausibility" that ingested hexavalent chromium can

cause GI cancer. The statement that he is not depending solely

on the Borneff study may literally be true. For example, Hattis

cites the California EPA reports. However, the fact is those

reports relied on the Borneff study to arrive at the risk figure

of 0.42 mg/kg-day. Furthermore, the Borneff study is a component

of each of the three risk co-efficients generated by Hattis.

      (b)   The Inquiry- "More Likely Than Not" or "Capable of

             Causing?"

It is important to determine exactly what analysis or

opinion of Hattis is at issue here in terms of scientific

reliability. Is it his **risk** co-efficients, in particular his

third co-efficient which results in an excess of 21 cancers?

"Risk" is what is analyzed in determining whether an agent is a

"more likely than not" cause of a particular disease. Or are

plaintiffs relying on Hattis merely as support for the

proposition that ingested hexavalent chromium is "capable of

causing" GI cancer? That, of course, is what the plaintiffs say

constitutes their "generic" causation burden of proof.

It appears plaintiffs assert they are relying on Hattis only

ORDER RE SUMMARY JUDGMENT-   **313**

1 for the proposition that Cr VI is "capable of causing" GI cancer,

2 and that his opinion in that regard is scientifically reliable.

3 According to plaintiffs, Hattis relies on "animal studies,

4 epidemiological evidence, distinguished regulatory committees and

5 as a Ph.D. in Genetics, his own expertise in the mechanisms of

6 cancer" to conclude ingested CR VI is carcinogenic. The question

7 therefore, is whether Hattis' opinion is scientifically reliable

8 for this more general proposition.

9

10   (i) **Epidemiological Studies**

11   Plaintiffs contend reliability is evident because Hattis

12 "analyzed the data of GI tract cancers from 3 epidemiological

13 studies of (humans) involving **occupational exposures to**

14 **chromium.**" However, these three studies involved **inhalation**

15 exposures. Hattis suggested that one way to compare ingestion

16 potency of Cr VI versus inhalation potency was to assemble all

17 the epidemiological studies in which respiratory and digestive

18 tumors have been observed in people and compare the apparent

19 excess of tumors over background for the two sets of sites.

20 (Hattis 1996 Rpt. at p. 29). According to Hattis:

21   The rationale for this is that **inhalation**
             exposures to mixed particulates of larger
22   sizes (over 1 micron) can be expected to
             include substantial gastrointestinal exposure
23   as particles deposited in the upper regions
             of the respiratory system are eventually swallowed
24   after being trapped and transported via the muco-
             cilliary escalator.

25 (_Id_.)

26

27   All Hattis could offer was a "preliminary analysis" of the

28 **ORDER RE SUMMARY JUDGMENT-   314**

1   three epidemiological studies, and he explicitly cautioned that

2   "[t]here is reason for concern that this preliminary result may

3   not be sustained at the same level in a more comprehensive

4   examination of the epidemiological literature." (Id. at p. 30).

5   As such, these studies are of no support for the proposition that

6   **ingested** Cr VI is "capable of causing" GI cancer. The simple

7   fact is there are no epidemiological studies finding a causal

8   association between Cr VI and GI cancer. Plaintiffs concede as

9   much.

10

11   **(ii)   Animal Studies**

12   In the absence of epidemiological studies, plaintiffs defend

13   Hattis' reliance on animal studies for the proposition that

14   **ingested** Cr VI is "capable of causing" GI cancer. The defendants

15   contend it is not scientifically proper for Hattis to rely on

16   animal studies alone. Defendants seemingly assert that case law

17   holds it is **never** scientifically proper to rely on animal studies

18   in the absence of epidemiological studies. However, the cases

19   cited by defendants clearly do not stand for such a per se

20   prohibition. Rather, the issue boils down to whether it is

21   scientifically proper for the expert to extrapolate from the

22   **particular** animal studies in question to arrive at conclusions

23   about health effects in humans.

24   The U.S. Supreme Court recognized this in its recent holding

25   in General Electric Co. v. Joiner, 118 S.Ct. 512 (1997). In

26   Joiner, the district court agreed with petitioners that the

27   animal studies on which the respondent's experts relied did not

28   **ORDER RE SUMMARY JUDGMENT-   315**

1 support his contention that exposure to PCBs had contributed to

2 his cancer.   The Supreme Court noted that respondent failed to

3 reply to this criticism:

> Rather than explaining how and why the
> experts could have extrapolated their
> opinions from these seemingly far-removed
> animal studies, respondent chose "to proceed
> **as if the only issue [was] whether animal
> studies can ever be a proper foundation for
> an expert's opinion."** [citation omitted].
> **Of course, whether animal studies can ever
> be a proper foundation for an expert's opinion
> was not the issue. The issue was whether
> these experts' opinions were sufficiently
> supported by the animal studies on which
> they purported to rely.** The studies were
> so dissimilar to the facts presented in
> this litigation that it was not an abuse
> of discretion for the District Court to
> have rejected the experts' reliance on them.

13 <u>Id</u>. at 518.   (Emphasis added).

14   The circuit courts view the issue the same way.   In <u>Raynor</u>

15 <u>v. Merrell Pharmaceuticals Inc.</u>, 104 F.3d 1371, 1376 (D.C. Cir.

16 1997), the court reiterated one of its prior holdings that <u>in</u>

17 <u>vitro</u>[232], <u>in vivo</u>[233], and chemical studies cannot, singly or in

18 combination, prove causation in human beings **in the face of an**

19 **overwhelming body of contradictory epidemiological evidence.**   In

20 <u>Allen v. Pennsylvania Engineering Corp.</u>, 102 F.3d 194 (5th Cir.

21 1996), the court stated that studies of the effects of chemicals

22 on animals must be carefully qualified in order to have

23 explanatory potential for human beings.   In <u>Allen</u>, none of the

24 scientific data on which the plaintiffs' experts relied furnished

25 ――――――――――――――

26 [232]   Experimentation on animal embryos.

27 [233]   Experimentation on live animals.

28 **ORDER RE SUMMARY JUDGMENT**-   316

1 a scientifically valid basis for the conclusions they sought to

2 draw. The paucity of epidemiological evidence, the unreliability

3 of animal studies, and the inconclusiveness of cell biology

4 combined to undercut the expert testimony. <u>Id</u>. at 198.

5   In <u>In re Paoli Railroad Yard PCB Litigation</u>, 35 F.3d 717

6 (3rd Cir. 1994), the Third Circuit found the district court had

7 abused its discretion in excluding the particular animal studies

8 at issue. The circuit distinguished other cases in which animal

9 studies had been found inadmissible due to extensive

10 epidemiological data failing to support causation, none of the

11 studies involved animals particularly similar to humans in terms

12 of reaction to the chemical in question, and none of the studies

13 had been relied upon by the federal government as a basis for

14 concluding the chemical was a probable health hazard. <u>Id</u>. at

15 780.

16   The probative value of animal studies also depends on the

17 nature of the causation inquiry: 1) is the agent a possible

18 cause of the disease ("capable of causing" the disease)?; or 2)

19 is the agent the probable or "more likely than not" cause of the

20 disease? In <u>Turpin v. Merrell Dow Pharmaceuticals, Inc.</u>, 959

21 F.2d 1349, 1359 (6th Cir. 1992), the court found the decisive

22 weakness in the plaintiffs' animal studies was that the factual

23 and theoretical bases articulated for the scientific opinions

24 stated would not support a finding that Bendectin **more probably**

25 **than not** caused the birth defects at issue. Plaintiffs' experts

26 testified the animal studies showed Bendectin was "capable of

27 causing," "could cause," or its effects were "consistent with

28 **ORDER RE SUMMARY JUDGMENT-   317**

1 causing" birth defects, not that it probably caused birth defects

2 in general or that it did in the particular case. Id. at 1360.

3 This was not enough. According to the court:

> We do not mean to intimate that animal studies
> lack scientific merit or power when it comes to
> predicting outcomes in humans. Animal studies
> often comprise the backbone of evidence indicating
> biological hazards, and their legal value has
> been recognized by federal courts and agencies.
> . . .
>
> **Here, the record's explanation of the animal studies
> is simply inadequate. Although the animal studies
> themselves may have been scientifically performed,
> the exact nature of these tests is explained only
> in general terms. The record fails to make clear
> why the varying doses of Bendectin or doxyalamine
> succinate given to rats, rabbits and in vitro animal
> cells would permit a jury to conclude that Bendectin
> more probably than not causes limb defects in children
> born to mothers who ingested the drug at prescribed
> doses during pregnancy. The analytical gap between
> the evidence presented and the inferences to be drawn
> on the ultimate issue of human birth defects is too
> wide.**

Id. (Emphasis added).

In Viterbo v. Dow Chemical Co., 826 F.2d 420 (5th Cir.

1987), plaintiff's expert relied on a study of rats showing that

when exposed to large amounts of a certain chemical, the rats

developed cancerous tumors and died. He admitted the effects of

chemicals differed between humans and rats. There was no

evidence the plaintiff had been exposed to comparable amounts,

nor that his symptoms were similar in any respect. According to

the court, the rat study, at most, was evidence the chemical

produced some unidentified effect on humans. It clearly was not

sufficient to support an opinion that the chemical caused the

plaintiff's depression, nervousness, hypertension, renal failure,

ORDER RE SUMMARY JUDGMENT-   318

1   and other ailments. Id. at 424.[234]

2   Based on the foregoing caselaw, the question is whether it

3   is scientifically proper for Dr. Hattis to extrapolate from the

4   Borneff **mouse** study to opine that ingested Cr VI is "capable of

5   causing" GI cancer in **humans**. Unlike Raynor, this is not a case

6   where there is overwhelming epidemiological evidence (human data)

7   that ingested Cr VI does **not** cause GI cancer in humans. If there

8   was such evidence, the court would probably be justified in

9   summarily dismissing Hattis' reliance on the mouse study.

10   However, there is simply no epidemiological evidence one way or

11   another whether ingested Cr VI is "capable of causing" GI cancer.

12   It is clear from the discussion above that the Borneff mouse

13   study is not scientifically reliable for the proposition that

14   ingested Cr VI is a "more likely than not" cause of GI cancer in

15   any members of the downwinder population. Hattis concedes this

16   by referring to his risk co-efficients as no more than a series

17   of "not clearly incorrect answers." (Hattis Dep. at p. 204).

18   However, there are also very serious questions whether the mouse

19   study is scientifically reliable for the proposition that

20   ingested Cr VI is "capable of causing" GI cancer in humans.

21   First of all, there is no escaping the fact that the Borneff

22   study did not even find a causal connection between ingested Cr

23

24   [234] In Hall v. Baxter Healthcare Corp., 947 F.Supp. 1387,
     1410 (D. Or. 1996), involving breast implant litigation, the
25   district court cited both Viterbo and Turpin for the proposition
     that "[e]xtrapolations of animal studies to humans are **generally**
26   not considered reliable in the absence of a scientific
     explanation of why such extrapolation is warranted." (Emphasis
27   added).

28   **ORDER RE SUMMARY JUDGMENT-**   **319**

1 VI and stomach cancer in **mice**. Hattis would have a stronger

2 argument if the Borneff study had come to a different conclusion.

3 On top of that, defendants point out a number of limitations in

4 the Borneff study, which are not rebutted by the plaintiffs: 1)

5 the mice consumed doses of potassium chromate (8.4 mg/kg-day)

6 significantly greater than Hattis' aggregate population dose

7 (1.56 mg/kg-day); 2) nearly two-thirds of the mice died during

8 the study due to "mouse pox;" 3) the results combined benign

9 forestomach tumors (papillomas) with non-tumor lesions

10 (hyperkeratomas) into one "benign" category; and 4) the observed

11 increase in forestomach lesions was confined almost exclusively

12 to the first generation of mice.

13   Furthermore, Hattis never offers an explanation why it is

14 scientifically defensible to compare mice to rats, and in turn

15 mice and rats ("rodents") to human beings in terms of cancer

16 potency (i.e. to believe ingested Cr VI would cause GI cancer in

17 humans **if** it caused a similar condition in rodents). He simply

18 "assumes" his 1/40 rodent ingestion to rodent inhalation potency

19 ratio is "indicative of [the] likely ratio of ingestion to

20 inhalation potency in people." As defendants point out, Hattis

21 does not cite any objective source supporting the potency ratio

22 employed by him.[235]

23   Considering all of these factors **together**, the court finds

24 _____

25   [235] This is not to suggest the use of ratios is
    inappropriate in comparing the potency of an agent in animals and
26 humans. However, the point is that a scientifically defensible
    basis for the comparison must be undertaken **before** a potency
27 ratio can even be justified.

28 **ORDER RE SUMMARY JUDGMENT-   320**

1 too great an analytical gap between the animal studies used by

2 Hattis (Borneff mouse ingestion study and the Glaser rat

3 inhalation study) and the inference sought to be drawn: that

4 ingested Cr VI is "capable of causing" GI cancer in humans.

5

6   **(iii) Regulatory Findings**

7   The findings of the California EPA workgroup are not

8 sufficient to bridge the analytical gap, particularly since the

9 workgroup also relied on the Borneff mouse study in recommending

10 ingested Cr IV be considered carcinogenic in humans. The

11 plaintiffs place great reliance on these regulatory findings.

12   In <u>Allen v. Pennsylvania Engineering Corp.</u>, the Fifth

13 Circuit observed:

14   Regulatory and advisory bodies such as IARC,
           OSHA and EPA[236] utilize a "weight of the evidence"
15   method to assess the carcinogenicity of various
           substances in human beings and suggest or make
16   prophylactic rules governing human exposure.
           This methodology results from the preventive
17   perspective that the agencies adopt in order to
           reduce public exposure to harmful substances.
18   The agencies' threshold of proof is reasonably
           lower than that appropriate in tort law, which
19   "traditionally make[s] more particularized
           inquiries into cause and effect" and requires
20   a plaintiff to prove "that it is more likely
           than not that another individual has caused
21   him or her harm." [Citation omitted].

22 102 F.2d at 198.

23   Plaintiffs seek to distinguish <u>Allen</u> on the basis that they

24 are relying on the findings of the California EPA workgroup only

25 _____

26   [236] Respectively, the International Agency for Research on
   Cancer, Occupational Safety and Health Administration, and
   Environmental Protection Agency.

27

28 **ORDER RE SUMMARY JUDGMENT-   321**

1 for the proposition that ingested Cr VI is "capable of causing"

2 GI cancer in humans, not that it is a "more likely than not"

3 cause of their diseases. This is immaterial.

4   Whether an agent is "capable of causing" a disease for

5 purposes of determining whether tort liability should be imposed

6 upon someone is still a wholly different proposition than whether

7 the agent should be considered carcinogenic as a regulatory

8 matter. Secondly, the California EPA committee relied on the

9 Borneff mouse study. For reasons set forth above, that study is

10 not even scientifically reliable for the proposition that

11 ingested Cr IV is "capable of causing" GI cancer in humans.

12   As defendants point out, the California EPA workgroup, in

13 order to justify its **recommendation**, considered both the "benign"

14 and "malignant" categories reported in Borneff and arrived at a

15 statistically significant result (in 66 female mice dosed with

16 potassium chromate, there were two malignant tumors and nine

17 benign tumors for a total of eleven; in 79 control female mice,

18 there were only two benign tumors). Hattis says this "reflects

19 an interpretation that in general benign tumors are likely to be

20 part of the multi-stage process that eventually can lead to

21 malignant tumors." (Hattis Affidavit at p. 27; Foulds Ex. 192).

22 While that may be considered persuasive evidence by a regulatory

23 body considering prophylactic rules, it is not reliable evidence

24 that ingested Cr VI is "capable of causing" GI cancer in humans.

25 This is particularly so where the evidence is derived from a

26 study in which the researchers did not distinguish between benign

27 tumors (papillomas) and benign lesions (hyperkeratomas), and

28 **ORDER RE SUMMARY JUDGMENT-   322**

1 ultimately concluded their study did not establish a causal link

2 between ingested Cr VI and stomach cancer in mice.[237]

3

4   (iv) **Biological Plausibility**

5   Lastly, plaintiffs assert Hattis has articulated a

6 biologically plausible scenario for how ingested Cr VI could

7 cause GI cancer in humans. At his deposition, Hattis indicated

8 that "acting by a DNA involved mechanism, . . . some amount of

9 chromium VI will probably get into epithelial cells of the lining

10 of the stomach and it will have some carcinogenic activity very

11 likely." (Hattis Dep. at p. 202). It does not appear defendants

12 specifically take issue with Hattis' theory of biological

13 plausibility.[238] However, "biological plausibility" is not

14 enough to save Hattis' opinion, particularly where there are no

15 epidemiological studies or animal studies reporting a statistical

16 association between ingested Cr VI and stomach cancer.[239]

17

18 [237] The plaintiffs cite a portion of the Borneff study in
which the researchers stated it was "suspected" with a
sufficiently long administration of a sufficiently high chromate

19 dose, it was "possible" that stomach carcinomas could appear in
mice. (Borneff, et al. 1968 at pp. 9-10; Defendants' Ex. 154).

20 Obviously, this does not make the study any more conclusive as to
whether ingested Cr VI is "capable of causing" stomach cancer in

21 mice.

22 [238] They do note that in the Borneff study, the researchers
reported tumors on the "forestomachs" on mice and that no

23 evidence has been presented that this is biologically comparable
to a human stomach.

24
[239] "Biological plausibility" is but one element of Hill's

25 criteria for inferring generic causal association. "Strength of
association" is another criteria established through relative

26 risk estimates. The association must be strong enough in order
to infer causal association. If the association is not strong

27 enough, "biological plausibility" is of no consequence. There

28 **ORDER RE SUMMARY JUDGMENT-**   **323**

1   **(c)**   **<u>Daubert</u> Criteria**

2   It appears Hattis' risk co-efficients were generated solely

3 for the purpose of this litigation. There is no indication that

4 prior to this litigation, or outside of this litigation, he

5 conducted any independent research concerning hexavalent chromium

6 and its effects on humans (particularly via the ingestion route).

7 Secondly, there is no indication Hattis' risk co-efficients and

8 the methodology which produced them have been peer reviewed or

9 published. The two principal ways for showing evidence satisfies

10 the reliability prong of <u>Daubert</u> is if it grows out of pre-

11 litigation research, or if the research has been subjected to

12 peer review. <u>Daubert II</u>, 43 F.3d at 1318.

13   Furthermore, there is no indication it is "generally

14 accepted" in the scientific community that ingestion of Cr VI is

15 "capable of causing" cancer in humans. The "recommendation" of

16 the California EPA committee that ingested Cr VI be considered

17 carcinogenic hardly amounts to "general acceptance." This

18 recommendation has not even been adopted by the California EPA.

19 Plaintiffs do not dispute that ATSDR has not classified ingested

20 Cr VI as carcinogenic, nor has the U.S. Environmental Protection

21 Agency or the IARC. (Rodricks Rpt. at pp. 17 and 32).[240]

22 _____

23 **must** be some degree of statistical association. Thompson, <u>Causal</u>
<u>Inference in Epidemiology: Implications For Toxic Tort</u>
<u>Litigation</u>, 71 N.C. L. Rev. 247, 266 and 269 (1992).

24

25   [240] The plaintiffs contend this is not significant since the
U.S. EPA has not even evaluated whether ingested Cr VI is
carcinogenic. EPA's failure to evaluate the carcinogenic

26 potential of ingested Cr VI is of no probative value to the
plaintiffs. It does not prove in the slightest that ingested Cr

27 VI is "capable of causing" cancer in humans.

28 **ORDER RE SUMMARY JUDGMENT-**   **324**

1   When these factors are considered in conjunction with the

2   methodological shortcomings identified above, it leaves the court

3   with no choice but to find Hattis' analysis, including his risk

4   co-efficients, unreliable for the proposition that ingested Cr VI

5   is "capable of causing" GI cancer in humans. In turn, there can

6   be no doubt his analysis is not scientifically reliable for the

7   proposition that ingested Cr VI is a "more likely than not" cause

8   of **any** of the plaintiffs' GI cancers.

9

10   c.   **Conclusion**

11   The court will grant defendants' motions in limine regarding

12   Drs. Hattis and Katz. Katz' report is irrelevant to the

13   determination of whether ingested Cr VI is a "more likely than

14   not" cause of any plaintiff's' claimed health effects-

15   carcinogenic and non-carcinogenic. Indeed, it is even irrelevant

16   to the determination of whether ingested Cr VI is "capable of

17   causing" any health effects. To the extent, if any, the report

18   purports to opine ingested Cr VI is "capable of causing" certain

19   health effects in humans, it is scientifically unreliable.

20   In his report, defense expert Rodricks appears to concede

21   ingested Cr VI is "capable of causing" non-carcinogenic toxicity.

22   His report assesses the potential for non-carcinogenic risk,

23   whereas he concludes "the current evidence available on

24   gastrointestinal cancers and exposure to hexavalent chromium is

25   clearly insufficient to establish causation." (Rodricks Rpt. at

26

27

28   **ORDER RE SUMMARY JUDGMENT-   325**

p. 29).[241] Nonetheless, the **plaintiffs** have not produced any

risk assessment regarding non-carcinogenic toxicity. Katz does

not discuss risk. Hattis limits his discussion of risk to GI

cancer. Accordingly, plaintiffs have no evidence from which a

jury could reasonably infer it is "more likely than not" that any

plaintiff's non-carcinogenic toxicity was due to hexavalent

chromium exposure from the Columbia River.

Hattis' 1996 report, to the extent it addresses Cr VI and GI

cancer, is irrelevant because it does not assist a jury in

determining whether it is "more likely than not" that any

plaintiff's GI cancer is due to hexavalent chromium exposure.

His report is scientifically unreliable for that proposition, as

well as for the proposition that ingested Cr VI is "capable of

causing" GI cancer.

Striking the Katz and Hattis reports on Daubert grounds

compels granting summary judgment for defendants on **all**

plaintiffs' health effects claims which are premised on alleged

exposure to hexavalent chromium emissions to the Columbia River.

2. Radionuclides

When reactor cooling water was returned to the river, it

also contained a quantity of non-iodine radioactive materials

including neptunium, sodium, zinc, arsenic and phosphorous.

According to Dr. Hattis, the purpose of his March 1996

---

[241] Rodricks concludes that historical exposures to
hexavalent chromium in the Columbia River water should not have
posed a health risk for local populations. (Rodricks Rpt. at p.
29).

ORDER RE SUMMARY JUDGMENT-   326

1 report ("Assessment of Population Aggregate Cancer Risks From

2 Radionuclides and Chromate Released by Hanford Operations into

3 the Columbia River") was to "supplement" HEDR's analysis of river

4 radionuclide emissions with: 1) census data on population sizes

5 for communities located along the Columbia River between 1950 and

6 1970; 2) estimates of the likely population variability of fish-

7 related exposures in general and an analysis of the distribution

8 of reported fish consumption rates for members of four Native

9 American tribes in the Columbia River basin; and 3) calculations

10 from HEDR data of the adjustments to mean dose estimates "needed

11 in the light of the likely uncertainty in one key set of

12 parameters"- the bioconcentration factors; and 4) estimates of

13 the cancer risk in relation to total equivalent whole body dose

14 from the radionuclides. (Hattis 1996 Rpt. at pp. 3-4).[242]

15   Because Hattis' report is intended to "supplement" HEDR's

16 analysis of river emissions, the logical place to start is with a

17 description of HEDR's analysis. The defendants have provided

18 such a description (based on the HEDR River Report[243]), with

19 which the plaintiffs do not take issue.

20

21   **a. HEDR Analysis of River Radionuclide Emissions**

22   HEDR (Hanford Environmental Dose Reconstruction Project)

23 _____

24   [242] In March 1997, Hattis submitted a "supplemental report."
    This will be discussed _infra_.

25

        [243] The HEDR River Report is officially known as Farris, et
26 al., (1994), _Columbia River Pathway Dosimetry Report, 1944-1992_.
    Defendants' Ex. 138. It will be referred to throughout as the
27 "River Report."

28 **ORDER RE SUMMARY JUDGMENT-   327**

1 analyzed the radionuclides released to Columbia River, quantified

2 their concentration at downstream locations, and developed a

3 computer model for estimating the radiation doses **potentially**

4 received by persons who drank water from the river, ate food

5 affected by the river, or used the river for recreational or

6 occupational purposes. HEDR divided the area of the Columbia

7 River downstream of Hanford into twelve segments beginning with

8 Ringold (Segment 1) and ending at the mouth of the Columbia River

9 (Segment 12).

10 There are three steps in the HEDR analysis. First, HEDR

11 developed a "source term" computer model to estimate the quantity

12 of radionuclides released to the Columbia River over particular

13 time periods. This model was constructed by analyzing the

14 physics and chemistry of Hanford reactor operations and by

15 reviewing original **operational records and historical**

16 **measurements** of radionuclides in Hanford reactor effluent.

17 Secondly, HEDR plotted the movement of these radionuclides

18 in the river and their concentration levels by using a computer

19 program that generated monthly estimates of concentrations at

20 downstream locations. The model simulated river flow and

21 transport, taking into account factors such as dilution,

22 radioactive decay, water volume, and flow rates on concentration

23 levels. HEDR also analyzed the concentration of radionuclides in

24 aquatic organisms, including fish, waterfowl, and Willipa Bay

25 oysters that lived in, or fed on, the Columbia River. The

26 analysis relied heavily on **historical measurements** collected by

27 Hanford researchers, universities and various state and federal

28 **ORDER RE SUMMARY JUDGMENT-   328**

1   agencies.

2   Finally, HEDR developed a computer model for estimating the

3   doses a person **could** have received at any of the twelve river

4   segments.   HEDR can compute individual doses based on information

5   such as the extent to which the individual consumed water or fish

6   from the river.   HEDR calculates and reports dose estimates for

7   several categories of hypothetical individuals who fit specific

8   dietary and lifestyle criteria "representative" of persons who

9   used the river.   It provides this **hypothetical** dose information

10 for all twelve river segments.

11   Two of the categories for which HEDR provides dose estimates

12 are the "typical representative individual" and the "maximum

13 representative individual."   The "typical representative

14 individual" is a hypothetical person who drank river water, but

15 ate no resident fish or waterfowl.[244]   HEDR's dose calculations

16 assume this person on an annual basis drank 444 liters of treated

17 river water, spent 25 hours in recreation on the river, and swam

18 for 12 hours in the river.   (HEDR River Rpt. at p. 3.27, Table

19 3.5).

20   The "maximum representative individual" is a hypothetical

21 person who consumed large quantities of fish, waterfowl and river

22 water.   HEDR's dose calculations assume this person annually ate

23 90 pounds of resident fish and 44 pounds of waterfowl, drank 740

24 liters of river water (8 of which were untreated), spent 504

25 _____

26   [244] Dose estimates for the "typical representative
    individual" were provided for all twelve river segments, even
    though not all communities along the river received their
27 drinking water from the river.

28 **ORDER RE SUMMARY JUDGMENT-   329**

1 hours of recreation on the river, and swam for 40 hours in the

2 river.   (HEDR River Rpt. at p. 3.26, Table 3.4).

3   The highest annual dose received by a "typical

4 representative individual" in any segment was 5 millirem EDE

5 ("effective dose equivalent"), reported for Segment 3 (Pasco and

6 Kennewick) for the years 1957, 1958 and 1960.   (HEDR River Rpt.

7 at Table A.4, Appendix A).   The highest annual dose received by a

8 "maximum representative individual" in any segment was 140

9 millirem EDE, reported for Segment 1 (Ringold) for the year 1960.

10 (HEDR River Rpt. at Table A.1, Appendix A).   Insofar as

11 cumulative dose estimates for the period 1950-1971, defendants

12 say the range was from a low of 16 millirem EDE for a "typical

13 representative individual" in Segment 12 ("Lower River" including

14 Portland and Vancouver), to a high of 1,531 millirem EDE for a

15 "maximum representative individual" in Segment 1 (Ringold).[245]

16

17   b.   **Hattis' Population Risk Analysis- 1996 Report**

18   Hattis assumes that all 596,000 persons he estimates lived

19 in communities along the Columbia River between 1950 and 1971

20 received radiation doses from the river.[246]   He set out to

21 _____

22   [245]   In his report, Hattis appears to say 18 millirem for the
    "typical representative individual" at Segment 12 (Hattis 1996
    Rpt. at p. 6), while Table A.4 of the HEDR River Report says 15

23 millirem.   The discrepancies are small and inconsequential for
    this motion.   Hattis reports a cumulative dose of 1,531 millirem

24 for Segment 1, however Table A.1 of the HEDR River Report shows a
    cumulative dose of 1,420 millirem.   The court is not sure what

25 accounts for this discrepancy, but it is also inconsequential.

26   [246]   The HEDR River Report covers the period from 1944 to
    1992 and Hattis' aggregate dose likewise takes into account that

27 entire period.   However, Hattis' total population figure- 596,000

28 **ORDER RE SUMMARY JUDGMENT-   330**

1   generate a "plausible" estimate of the number of excess cancers

2   that could occur in the population as a result of the assumed

3   radiation exposure.   (Hattis October 1997 Affidavit at p. 1,

4   Paragraph 2, Ex. 4 to Plaintiffs' Appendix 1 re Non-Iodine

5   Claims, hereinafter "Hattis Affidavit").   There are two variables

6   in Hattis' population risk analysis: 1) a population dose which

7   is the sum of the radiation doses received by all members of the

8   assumed exposed population; and 2) a risk co-efficient (the

9   measure of the risk of cancer per unit of radiation dose).   The

10   population dose is multiplied by the risk co-efficient to yield

11   the number of excess cancers predicted for the population.

12   Hattis generated alternative estimates of population dose

13   which are expressed in terms of "person-rem."   The first set of

14   estimates assumes that in each of the twelve river segments,

15   either 1% or 5% of the assumed exposed population received at

16   least the same dose HEDR estimated for its "maximum

17   representative individual."   (Hattis 1996 Rpt. at p. 15, Table

18   4).   The doses are expressed in terms of 99th percentile (1%

19   received such a dose) and 95th percentile (5% received such a

20   dose).   The cumulative 99th percentile dose is 36,900 rem

21   (3.69E+04) and the cumulative 95th percentile dose is 101,000 rem

22   (1.01E+05).   Hattis then **averaged** these doses over his entire

23   exposed population.   They translate into respective **average**

24   _____

25   -is an average derived from census data for the period **1950-1971**.
     Likewise, HEDR's "Summary of Estimated Columbia River Doses"
26   (Appendix A) lists annual and cumulative doses for the period of
     1950 to 1971, roughly corresponding to the period during which
27   the "river-cooled" reactors were in operation.

28   **ORDER RE SUMMARY JUDGMENT-   331**

1 **cumulative** doses per person of .0619 rem or 62 millirem

2 (36,900/596,000) and .169 rem or 169 millirem (101,000/596,000).

3   Hattis then generated a second set of estimates which made

4 the same assumption, but increased HEDR's reported dose estimates

5 for the maximum representative individual by 64% (1.64) or 98%

6 (1.98), depending on the river segment (98% for Segments 1-6; 64%

7 for Segments 7-12). The increase is to account for a

8 "correction" Hattis makes to HEDR's treatment of bioconcentration

9 factors. (Hattis 1996 Rpt. at p. 23). Based on their

10 calculations, defendants say the 99th percentile cumulative dose

11 increases to 52,999 rem, while the 95th percentile dose increases

12 to 208,500 rem. The average cumulative doses per person increase

13 to 88 millirem or .088 rem (52,999/596,000) and 349 millirem or

14 .349 rem (208,500/596,000) respectively. Plaintiffs do not

15 dispute these calculations.

16   These population dose estimates are then multiplied by a

17 risk co-efficient to yield the number of excess cancers predicted

18 for the population. According to defendants, for his population

19 risk computation, Hattis uses a risk co-efficient which lumps all

20 of the cancer categories together and generates a single estimate

21 for all cancers.[247] Table 8 of Hattis' report (p. 22) contains

22 his prediction of the excess number of cancers (deaths and cases)

23 based on the 99th and 95th percentile doses, without "correction"

24 of the bioconcentration factor. Table 10 (p. 25) contains his

25 _____

26   [247] The defendants do not precisely describe how these
calculations work, but once again, plaintiffs do not dispute the
27 defendants' description of Hattis' analysis.

28 **ORDER RE SUMMARY JUDGMENT-**   **332**

1 prediction with "correction" of the bioconcentration factor.

2 Each table contains alternative assumptions about risk.

3   For his "baseline" risk co-efficients, Hattis uses what he

4 refers to as the "official" risk estimates of the United States

5 Environmental Protection Agency (EPA). (Hattis 1996 Rpt. at p.

6 18, Table 5). Because a "prudent policy planner will want to

7 consider the potency estimates in Table 5 as the lower bounds of

8 an approximately 2-3 fold credible central range of likely

9 overall cancer risk," Hattis proposes alternative risk

10 assumptions increasing the risk co-efficient by a factor of 2 for

11 fatal cancers (cancer deaths) and by a factor of 3 for non-fatal

12 cancers (cancer cases). For example, in Table 10, the 99th

13 percentile dose for the "maximum representative individual"

14 generates an estimated number of 27 fatal cancers based on the

15 EPA risk co-efficient. Increased by a factor of 2, it is 54.

16 The estimated number of non-fatal cancers, based on the EPA risk

17 co-efficient, is 40. Increased by a factor of 3, it is 121.

18   Hattis concludes that a "plausible" range for the number of

19 total cancers" that could appear among the 596,000 persons

20 assumedly exposed to radiation from the Columbia River is 40-475,

21 including 27-212 fatal cancers. (See Table 10 at p. 25).

22

23   **(1) Fit/Relevancy of Population Risk Analysis-1996 Report**

24   Defendants contend Hattis' population risk analysis does not

25 "fit" the relevant causation inquiry. This, say defendants, is

26 because he does not opine that the radiation doses he attributes

27 to the Columbia River doubled **anyone's** risk of cancer.

28 **ORDER RE SUMMARY JUDGMENT-**   **333**

1   Defendants are quick to point out that Hattis employed the

2   "doubling of risk" standard in a 1995 report he prepared **outside**

3   of this litigation and entitled "Radiation-Induced Cancers in DOE

4   and Contractor Employees: Implications of Possible Alternative

5   Workers' Compensation Settlement Policies and Assessment of the

6   Possible Role of New Molecular Biological Techniques."[248] In

7   Table 15 of that report, Hattis listed "Doubling Doses for

8   Selected Cancer Sites for Low Dose Rate Ionizing Radiation

9   Exposure, Calculated From EPA Age Specific Risk Coefficients

10   Incorporating a Dose and Dose Rate Effectiveness Factor of 2."

11   (Hattis 1995 Rpt. at p. 40).[249]

12   Defendants note the HEDR River Report identifies the colon

13   (the lower large intestine) as the organ receiving the highest

14   river dose. (River Report at p. 5.4). For the "maximum

15   representative individual" at Ringold (Segment 1), the cumulative

16   organ dose reported is 5,070 millirem. (Table A.3 at p. A.9,

17   Appendix A to River Rpt.). This is the highest cumulative dose

18   reported for any of the twelve river segments. With Hattis'

19   bioconcentration factor adjustment of 1.98, the dose would be

20   10,038 millirem (5,070 x 1.98). Table 15 of Hattis' 1995 report

21   shows a doubling dose range for colon cancer of 234,000 millirem

22   (234 rem) to 1,112,000 millirem (1,112 rem), depending on the age

23   group. Obviously, that range of doses far exceeds 10,038

24   _____

25   [248] Referred to hereinafter as the "1995 Report."

26   [249] Apparently, the report was revised sometime in 1996.
     However, Table 15 remained in the report at p. 40. Defendants'
27   Ex. 45.

28   **ORDER RE SUMMARY JUDGMENT-   334**

1 millirem.   Indeed, the **lowest** doubling dose found on Table 15 of

2 Hattis' 1995 report is 31,000 millirem (31 rem) for leukemia,

3 which also exceeds 10,038 millirem.[250]

4   Defendants assert that Hattis' 1995 non-litigation report,

5 combined with his 1996 litigation report, "demonstrates that no

6 individual can prove causation at the radiation doses claimed

7 here."   The court is not convinced that is necessarily true since

8 Table 15 pertains to only three age groups: 20-29; 30-39 and 40

9 and over.   It does not provide any doubling dose figures for

10 individuals under age 20 at the time of exposure.   Actually, that

11 is not surprising since Hattis' 1995 report pertains to "DOE and

12 Contractor Employees" and therefore, presumably to "occupational"

13 exposures.   Individuals under age 20 probably comprise a very

14 small fraction of the workforce.   As has been discussed

15 elsewhere, the risks for some types of cancer (notably, thyroid

16 cancer) are greater in children and adolescents.

17   According to defendants, Hattis "retreats" to a population

18 risk analysis to avoid having to reconcile the river doses in his

19 1996 litigation report with the doubling doses found in Table 15

20 of his 1995 non-litigation report.   Indeed, in their response

21 brief, plaintiffs do not dispute defendants' contention that the

22 highest river organ dose derived from Hattis' calculations does

23 not exceed any of the doubling doses set forth in Table 15 of his

24 _____

25   [250] In his 1997 "supplemental report" and affidavit, Hattis
   increases his river doses, including his large lower intestine
26 doses, to levels which in some cases exceed the doubling doses
   set forth in Table 15 of his 1995 report.   The "supplemental"
27 report and affidavit are discussed _infra_.

28 **ORDER RE SUMMARY JUDGMENT-   335**

1   1995 report.   Nor do plaintiffs (or Hattis) contend the highest

2   river dose would exceed doubling doses for individuals under age

3   20, had those been presented by Hattis in Table 15 of his 1995

4   report.   In other words, plaintiffs do not suggest what the

5   doubling doses might be.

6   Instead, plaintiffs assert, once again, that "doubling dose"

7   is an individual causation standard which is irrelevant at this

8   "generic causation" stage of the proceedings.   They assert

9   Hattis' **population** risk analysis is relevant to and supports

10   their generic causation burden of proving radiation from the

11   Columbia River is "capable of causing" the cancers suffered by

12   them.[251]

13   Plaintiffs cannot dispute that the population risk analysis

14   _____

     [251] The defendants contend that while a properly performed
15   analysis of population risk might be of use to regulators charged
     with formulating public health policy, it is irrelevant in a
16   toxic tort case which demands each plaintiff prove that radiation
     exposure doubled his/her risk of contracting cancer.
17   In his 1996 report, Hattis refers to the "prudent policy
     planner" wanting to consider the EPA potency estimates as the
18   lower bounds of risk.   (Hattis 1996 Rpt. at p. 21).   In their
     response brief, plaintiffs quote from an NCRP publication that
19   the concept of "collective dose" has found "increasing
     application in radiation protection, both as an operational tool
20   for controlling radiation exposures to radiation workers and to
     the general public, and as a means for estimating the **prospective**
21   risks to populations from real or potential radiation exposures."
     (NCRP, NCRP Report No. 121:   Principles and Application of
22   Collective Dose in Radiation Protection (November 30, 1995) at p.
     61)(Emphasis added). (Defendants' Ex. 158).
23   These references by plaintiffs and their expert show that
     collective or population dose is geared toward gauging
24   prospective risk as a regulatory matter, not for determining the
     likelihood that radiation exposures are the legal cause of
25   existing cancers suffered by plaintiffs.   Indeed, according to
     defendants' expert, Dr. John Frazier, collective dose should not
26   be used for "retrospective assessments of potential past
     detriments or risks."   (Frazier 1996 Rpt. at p. 7).

27

28   **ORDER RE SUMMARY JUDGMENT-   336**

1   contained in **Hattis' 1996 report** does not opine that radiation

2   exposures from the river- "assumed" exposures in this case-

3   doubled **anyone's risk of cancer.**[252]   This is the "generic"

4   inquiry before the court.   It is an inquiry about the plaintiffs

5   as a **collective** unit.   It is not an inquiry pertaining to any

6   **particular** individual plaintiff.

7   Based on his **population** risk analysis, Hattis is unable to

8   state that any of his "predicted" cancers have occurred, or that

9   his "excess" includes anybody who might be a claimant in this

10   case.   (Hattis Dep. at p. 94;   Hattis 1996 Rpt. at p. 23

11   discussing "plausible" range of aggregate cancers). He has no

12   basis for saying that **any** plaintiff in this case, suffering from

13   cancer, can attribute that condition to radiation exposure from

14   the Columbia River.

15   Finally, defendants assert Hattis' analysis does not even

16   show a doubling of the risk at the **population** level.   While he

17   assumes 596,000 persons were exposed to radiation from the

18   Columbia River, his estimated or predicted range of excess

19   cancers is 40 to 475 for the entire population.   According to

20   defendants' calculations, this amounts to an increased risk of

21   less than 1/4 of 1 percent.   Defendants assume a background

22   incidence of cancer in the United States of 35%.   (Radford Dep.

23

24   _____

25   [252]   Hattis comes up with an "excess" number of cancers which
he claims are due to exposure to a dose of radiation from the
26   river received by HEDR's "maximum representative individual."
However, he does not offer an opinion as to the likelihood that
27   such a dose caused cancer in **any** such individuals.

28   **ORDER RE SUMMARY JUDGMENT-**   **337**

at pp. 45-46).[253] This means that in a population of 596,000,

208,600 people would be expected to get cancer in the normal

course (596,000 x 35%). An excess of 475 cancers due to assumed

radiation exposure from the river amounts to an increase of 0.227

percent (209,075/208,600).

The plaintiffs do not confront these arguments because they

contend it is irrelevant to their generic causation burden of

proof. Nonetheless, even if the plaintiffs can prove river

radiation exposure is "capable of causing" their cancers, it does

not raise an inference it is a "more likely than not" cause.

Consequently, a jury cannot sustain a verdict for **any** plaintiff

based on purported river exposures.

The court will exclude Hattis' 1996 report because it is

irrelevant to the generic causation inquiry before this court.

(2) Reliability of Population Risk Analysis-1996 Report

Defendants also take issue with the scientific reliability

of the population risk analysis set forth in Hattis' 1996 report.

The fundamental question is whether it is proper for Hattis

to use HEDR's river analysis for the purpose of performing a

population risk analysis. HEDR calculated its river doses for

hypothetical people (the "typical" and "maximum" representative

individual) in each of the 12 segments. For example, HEDR could

assume residents of each segment drank river water even if they

in fact did not. Hattis used HEDR's analysis to calculate a dose

---

[253] American Cancer Society data, cited elsewhere in
defendants' briefs, confirms this background figure.

ORDER RE SUMMARY JUDGMENT-   338

1 for the total population of **actual** people living in each of the

2 12 segments during the relevant time.

3   Hattis estimated the average dose at each river segment by

4 extrapolating from the dose estimates HEDR provided for its

5 **hypothetical** typical and maximum representative individuals. He

6 used a mathematical formula to calculate an average or **mean** value

7 in a distribution of numbers.   (Hattis Rpt. at 13 and n. 33).

8 The formula applies to numbers distributed in a manner called

9 "lognormal" and where two values are known: 1) the median value-

10 the middle (50th percentile) value in a distribution of numbers

11 and 2) the geometric standard deviation- a measure of the

12 variation between the mean value and individual values within a

13 set of data.   In a lognormal distribution, the values are

14 distributed in a "skewed" way such that the mean value is always

15 higher than the median value.[254]

16   According to defendants, Hattis made several unsubstantiated

17 assumptions in applying his formula, including: 1) he assumed

18 that for each river segment the median dose for his exposed

19 population was the same dose HEDR reported for its "typical

20 representative individual;" 2) to calculate his geometric

21 standard deviation, he assumed that for each river segment, the

22 _____

23 [254] This description, supplied by defendants, is not
disputed by the plaintiffs.

24   "Mean" is one way to find the center of a batch of numbers:
Add up the numbers, and divide by how many there are. "Median"
is another way to find the center of a batch of numbers. The

25 median is the fiftieth percentile. Half the numbers are larger,
and half are smaller. FJC Reference Manual on Scientific

26 Evidence, "Reference Guide on Statistics" at p. 400. The "mean"
varies in terms of percentile dose, while the "median" is always

27 a 50% dose.

28 **ORDER RE SUMMARY JUDGMENT-   339**

1 maximum dose received by any member of his exposed population was

2 the same dose HEDR reported for its "maximum representative

3 individual" and that either 1 percent or 5 percent of the

4 population received that dose; and 3) he assumed his exposed

5 population received radiation doses distributed in a lognormal

6 manner.

7

8   **(a)   Does Actual Exposed Population Fit the Criteria**

9   **on Which HEDR Bases Its Hypothetical Dose Estimates?**

10   Defendants contend the "threshold" problem with Hattis'

11 analysis is that he does not know how HEDR's doses for its

12 hypothetical "typical" and "maximum" representative individuals

13 compare with doses actually received by the 596,000 members of

14 his exposed population. They note that HEDR did not analyze

15 population risk and therefore, did not have any reason to

16 investigate the extent to which residents living in communities

17 along the Columbia River had the same characteristics as the

18 hypothetical individuals. HEDR did not investigate the range and

19 distribution of doses actually received by persons in those

20 communities. It merely provided a mechanism by which individuals

21 could estimate the dose received by them based on the

22 hypothetical profiles. Because Hattis **was** trying to analyze

23 population risk, defendants say it was necessary for him to have

24 reliable estimates of the doses that members of his exposed

25 population **actually** received.

26   The plaintiffs try to counter this argument as follows:

27   While defendants have been quick to admonish

28 **ORDER RE SUMMARY JUDGMENT-**   **340**

> plaintiffs' experts for engaging in 'speculation,'
> apparently it's permissible for them to rest
> their summary judgment motions on speculative dose
> estimates since it's Batelle who's doing the
> speculating. Defendants can't have it both ways.
> Either HEDR's 'representative' dose estimates are
> sufficiently concrete to support defendants' motion
> to dismiss all non-thyroid claims, or they are so
> 'hypothetical' to be of absolutely no value in this
> proceeding.

This is an effort to shift the burden of proof to the defendants. The defendants do not bear the burden of proving the range of doses **actually** received by **any** of the plaintiffs. The plaintiffs have this burden. Indeed, if plaintiffs were dissatisfied with HEDR, they had no obligation to use it. They had the option of producing their own model. The defendants have supplied what are effectively guidelines for determining **actual doses** received by individuals, but it is incumbent upon the **plaintiffs** to supply the **actual dose** information to plug into the guidelines.[255] This is precisely what the plaintiffs have not done. Instead, as will be discussed, plaintiffs and Dr. Hattis make unsubstantiated assumptions about water consumption, fish consumption, etc., among the "exposed" population as a whole (the 596,000 individuals).

Plaintiffs say the Hanford Health Effects Panel did not ask HEDR for "hypothetical" representative individual doses and that "[w]ithout the substantial resources at his disposal to conduct

---

[255] HEDR's guidelines allow an individual to roughly compute the type of dose he might have received from the river. However, by itself, it does not allow him to determine the risk that the estimated dose was the cause of his cancer. Additional information is necessary to make the risk assessment- i.e. epidemiological data from which risk co-efficients can be derived. (See discussion _infra_ re Hattis supplemental report).

ORDER RE SUMMARY JUDGMENT-   **341**

the extensive fish surveys demanded by the defendants, Dr. Hattis

extends HEDR's representative individual dose to calculate the

collective dose originally requested by the Hanford Health

Effects Panel." This argument essentially seems to be that

because HEDR allegedly did not do its job- did not calculate

actual doses- the plaintiffs do not have to do it either. It is

a tacit concession by plaintiffs that they do not have the type

of information (i.e. fish consumption data, water consumption

data, etc.) from which to derive reliable estimates of **actual**

dose received. Whether Batelle performed its political or

contractual mandate does not excuse plaintiffs' burden of proving

causation in a **tort** claim in a court of law.

          (b) "Typical Representative Individual" Same as "Average"

                Individual in Exposed Population?

     Hattis assumes that for each river segment, the median dose

of his exposed population (the **actual dose** received) is the same

dose HEDR reported for its "typical representative individual."

In his 1996 report, Hattis says it "appears" the "typical

representative individual" is a "median adult- 50% of **actual**

people would be likely to have greater exposures and 50% smaller

exposures." (Hattis 1996 Rpt. at p. 2). When asked at his

deposition whether he assumed HEDR's "typical representative

individual" meant a "median" individual, Hattis responded: "I

thought that's what they meant." (Hattis Dep. at pp. 100-

01)(Emphasis added).

     Defendants contend this is a wholly subjective assertion

ORDER RE SUMMARY JUDGMENT-   342

1 without any evidentiary support. Here again, observe the

2 defendants, HEDR did not analyze or provide information

3 concerning the distribution of **actual** doses for any population

4 along any river segment. Also, say defendants, HEDR did not

5 claim its "typical representative" dose was an estimate of the

6 "median" dose for persons living in communities near the Columbia

7 River. Rather, HEDR stated its representative individuals did

8 not have characteristics of the general population, but only of

9 "selected segments" thereof:

> The characteristics of these individuals are
> intended to approximate those of **selected
> segments** of the general population. **The
> characteristics of the representative individuals
> do not match any <u>known</u> person.** The representative
> individuals are used to estimate the doses to
> these selected population segments.

14 (River Rpt. at p. 3.25)(Emphasis added).

15   According to defendants, because Hattis did not investigate

16 the extent to which the members of his actual population fit

17 HEDR's definitions of "representative individuals", he has no

18 basis for saying his population's median dose is the same dose

19 HEDR reported for the "typical representative individual."

20   In response, plaintiffs tender the same argument as before.

21 They suggest the burden of proof should be shifted to the

22 defendants and essentially admit they do not have the information

23 to compute reliable actual dose estimates:

> HEDR never calculated the population dose it
> was asked to perform by the Hanford Health
> Effects Panel. Even though Batelle was saved
> the considerable expense of conducting surveys
> or obtaining specific fish consumption data from
> locations downstream, defendants now require that
> plaintiffs finance such an undertaking to support

28 **ORDER RE SUMMARY JUDGMENT-   343**

1   its experts' opinions on generic causation.

2   (Plaintiffs' Response Br. at p. 63).

3   Plaintiffs contend Hattis' "subjective judgments based on

4   his expertise warrant the same deference in the scientific

5   context that underlies Batelle's river modeling decision-making."

6   They cite a passage from Batelle's "Recommendation to Technical

7   Steering Panel Regarding Approach for Estimating Individual

8   Radiation Doses Resulting from Releases of Radionuclides to the

9   Columbia River:"

> Some of the estimates required judgments
> based on expert opinion. Judgments are an
> integral and necessary part of all decision
> modeling. For some of the inputs, objective
> data were available and used wherever possible.
> **Often objective data were available for baseline
> estimates at a particular time and/or place,
> which were then modified by judgment to fit
> the particular circumstances of other times or
> places prior to input.**
>
> The required judgments were provided by the
> authors and verified by other individuals with
> the **appropriate knowledge** who provided feedback,
> which led to consensus on the estimates.

18 (PNWD-1977 at p. 4.1, Foulds Ex. 229)(Emphasis added).

19   This passage makes clear that even "subjective" judgments,

20 in order to be considered scientifically reliable, must have some

21 basis in supporting "objective data," or must be verified by

22 someone with "appropriate knowledge." Hattis has no objective

23 data (fish consumption data, water consumption data, etc.) to

24 support his conclusion that the "median" doses received by his

25 actual exposed population are the same as the doses received by

26 HEDR's "typical representative individuals." Furthermore, no one

27 with "appropriate knowledge" has "verified" Hattis' subjective

28 **ORDER RE SUMMARY JUDGMENT-   344**

1 assertion.   As defendants point out, Hattis' analysis has not

2 been peer reviewed.[256]

3   Plaintiffs contend Hattis' use of HEDR's "typical individual

4 representative" dose as a "median" dose point for his exposed

5 population is reasonable because "typical" and "median" are

6 "average" by definition.   They cite HEDR's "River Report" which

7 defines "typical representative individual" as "typical of the

8 **average** individual residing near the Columbia River."   (River

9 Rpt. at p. 3.25).

10   The **"average"** population doses referred to in Hattis' report

11 are **not** "median" doses, but "mean" doses.   According to Hattis:

12 "[W]e need estimates of **population average (arithmetic mean)**

13 doses in order to calculate the number of extra cancer cases that

14 are expected to occur as a result of exposures."   (Hattis 1996

15 Rpt. at p. 2)(Emphasis added).   Hattis' mean ("average")

16 population doses are **higher** than his median doses which are based

17 on HEDR's "typical representative individual" doses.   (Id. at p.

18 13 and Table 3 at p. 14).   At his deposition, Hattis stated he

19 thought "typical" and "median" were synonymous, but he did not

20 assert that "median" and "average" were the same.

21   The plaintiffs argue defendants' Daubert motion is an

22 attempt to impose upon Hattis the defendants' choice of "numerous

23 available methodologies and assumptions of calculating population

24 dose."   That is not the case at all.   Rather, what the defendants

25 are saying is that HEDR's River Report was not intended to

26 _____

27   [256]   See discussion of Daubert criteria, infra.

28 **ORDER RE SUMMARY JUDGMENT-   345**

1 calculate population dose and Hattis cannot use it for that

2 purpose.   Nobody said Hattis had to use HEDR's River Report.

3 With sufficient and reliable data about **actual** consumption

4 practices along the Columbia River, perhaps a reliable population

5 dose estimate could be calculated from which there might be a

6 reliable assessment of population risk.[257]   Of course, even a

7 reliable population risk analysis does not help to determine

8 whether radiation exposure doubled the risk of **any** individual

9 getting cancer.

10 Plaintiffs assert Hattis' assumption about "average" dose is

11 justified because of a 1961 U.S. Public Health Survey

12 (Plaintiffs' Response Br. at pp. 67-70), but as defendants point

13 out, Hattis did not rely on this data in his report.   Because it

14 is not part of an expert analysis, it is not admissible on its

15 own.

16

17 **(c)   Use of "Maximum Representative Individual" for**

18 **Maximum Doses**

19 In calculating the geometric standard deviation required by

20 _____

[257]   Plaintiffs contend defendants "neglect" to point out
21 that HEDR's "typical" and "maximum" representative individuals
are assumed to have consumed certain quantities of salmon,
22 steelhead and shellfish (so-called "non-resident" fish).
According to plaintiffs, defendants' failure to point this out
23 may be due to a number of reasons, including that "these modest
salmon-steelhead-shellfish assumptions **may** drastically
24 underestimate **actual** consumption."   (Defendants' Response Br. at
p. 65)(Emphasis added).
25 It is plaintiffs' burden to prove if that is the case.
Plaintiffs are the ones who need to supply the **actual** consumption
26 data if they want to prove an **actual** population dose (or **actual**
individual dose) as opposed to a **hypothetical** population dose (or
27 **hypothetical** individual dose).

28 **ORDER RE SUMMARY JUDGMENT-   346**

his formula, defendants say Hattis made two assumptions: 1) he assumed for each river segment that the maximum dose received by any member of his exposed population was the same dose HEDR reported for its "maximum representative individual;" and 2) he assumed that either 1 percent or 5 percent of his exposed population had the same doses as HEDR's "maximum representative individual."

Defendants contend the first assumption fails for the same reason as the assumption that HEDR's "typical representative individual" dose equates to the "average" dose received by members of the exposed population: Hattis does not provide any evidentiary basis for the assumption and does not show that any member of his exposed population had dietary and lifestyle characteristics similar to HEDR's "maximum representative individual."

In his affidavit, Hattis states he did not assume that for each river segment the maximum dose any member received was the same dose HEDR reported for its "maximum representative individual." According to Hattis:

> I assumed that HEDR's 'maximum representative individual' corresponded to the dose rate for a high, but certainly not the highest, exposures in the population groups considered. This was in part because I had direct data that some members of the population (surveyed in the Columbia Intertribal Fish Commission Study[258]) consume a great deal more fish than HEDR's 'maximum' assumptions. As should have been clear to even a casual reader of my report, I made alternative calculations corresponding to alternative possibilities that 1% or

---

[258] Also referred to herein as the "Native American fish survey."

ORDER RE SUMMARY JUDGMENT-   347

5% of the population could have doses that **exceeded** HEDR's 'maximum representative individual' doses.

(Hattis Affidavit at pp. 4-5; Ex. 4 to Plaintiffs' Appendix 1 re Non-Iodine Claims)(Emphasis added).

Even if that is the case, the question still remains whether Hattis had data from which he could reliably conclude that 1% or 5% of his **entire** exposed population could have doses exceeding HEDR's "maximum representative individual" doses.

One of the sources of data he relied upon was a survey of members of four Native American tribes "located" in the Columbia River basin. (Hattis 1996 Rpt. at p. 11). This is the Columbia Intertribal Fish Commission Study, also referred to as the Native American fish survey. In his 1996 report, Hattis indicated over 90% of those surveyed reported eating some fish in "local areas, including the Columbia River." Based on equations performed by him, Hattis estimated that over 13% of fish eaters in the survey would have consumed **more** fish than HEDR's "maximum representative individual" (roughly in excess of 40 kg of all types of fish, both resident and non-resident). (Id. at p. 11).

Defendants contend this survey does not provide Hattis with what he needs to analyze the risk for his **entire** exposed population, which is "a reliable estimate, by river segment, of the number of persons within his mostly non-Native American exposed population who received the same [or greater] dose than HEDR's "maximum representative individual." According to defendants, Hattis does not: 1) show how the results of the survey could be extended to his mostly non-Native American

ORDER RE SUMMARY JUDGMENT-   **348**

exposed population and how it supports his assumption that 1

percent or 5 percent of the population at each river segment had

the same dose as HEDR's "maximum representative individual;" 2)

does not specify the number of Native Americans in the survey, if

any, who lived in communities along the Columbia River that are

included in his exposed population, nor identify the persons in

his exposed population to whom he believes the results of his

survey apply; 3) does not compare the species of fish consumed by

the reporting Native American population with the species HEDR

assumed were consumed by its "maximum representative

individual."[259]

Hattis acknowledged there were some limitations with this

survey. In his report, he stated:

> Of course, Native Americans of these four tribes
> do not constitute a majority of those who live
> on or near the Columbia River, and even the tribes
> in this survey do not do all of their fish harvesting
> in the Columbia River.

(Hattis 1996 Rpt. at pp. 11-12). Nonetheless, Hattis asserted

"there is known to be a **respectable** number of people in the

general U.S. population who have locally-caught fish as a major

portion of their diets." (Id. at p. 12)(Emphasis added). In

that regard, he cited a 1970 U.S Fish and Wildlife Survey[260]

---

[259] HEDR studied three types of Columbia River fish: 1)
omnivorous fish (bullhead, catfish, suckers, whitefish,
chiselmouth, chub, sturgeon, minnows, and shiners); 2) first-
order predators (perch, crappie, punkinseed, and bluegill); 3)
second-order predators (bass, trout, and squawfish). Salmon and
steelhead were treated separately. HEDR River Rpt. at p. 3.14.

[260] Fish and Wildlife Service, **National Survey of Fishing
and Hunting 1970** (1972). Defendants' Ex. 159.

ORDER RE SUMMARY JUDGMENT-   349

1 which he claims "estimated that there were about 750,000 people

2 in the U.S. who took over 100 fishing trips per year."   Added

3 Hattis:

4   This is a group of people-- somewhat under 0.5%
         of the population-- who can be **expected** to be
5   regular consumers of locally caught fish . . . .
         And they can also be **expected** to have provided
6   significant amounts of those fish to family members,
         suggesting a somewhat larger number of relatively
7   high intensity local fish consumers in the general
         population. Overall, considering the presence of
8   the Native American tribes in the Columbia River
         Basin added to the general population frequency
9   of subsistence fishers, in my judgment the 40kg fish
         consumption rate postulated for the 'maximum
10   representative individual' probably represents
         between a 95th and 99th percentile for those living
11   near the Columbia River.

12 (Hattis 1996 Rpt. at p. 12)(Emphasis added).

13   Obviously, the problem with this survey is it concerns the

14 **frequency of fishing trips** and says nothing about **fish**

15 **consumption**, or the amount or species of fish sport fishers

16 provide to family members. It also says nothing about the

17 Columbia River. It does not support Hattis' assumption that for

18 **each** Columbia river segment, 1 percent or 5 percent of his **entire**

19 exposed population (596,000 persons) had the same dietary and

20 lifestyle characteristics as HEDR's "maximum representative

21 individual."

22   In his affidavit, Hattis acknowledges the shortcomings of

23 the U.S. Fish and Wildlife Survey:

24   The actual population size involved is subject
         to modification based on data on how much of
25   what kind of fish was actually taken from the
         river by sport/subsistence fishers, and what
26   fraction of the population actually consumed
         fish from the river in the relevant time periods.
27   If I eventually give testimony, that testimony

28 **ORDER RE SUMMARY JUDGMENT-   350**

1   will reflect available information on those
         points that I have at that time.

2 (Hattis Affidavit at p. 3, Paragraph 9).

3   Elsewhere in his affidavit, Hattis states:

4   Data on total fish catch from the Columbia River,
         and the fraction of the Columbia River community
5   residents who ate no fish are relevant to the
         assessment of aggregate [population] dose. Also
6   relevant are the correspondence between the species
         of fish reported to be caught and consumed and the
7   seasons of the year when they are caught in relation
         to the assumptions made in the HEDR calculations. . . .
8   If I am asked to give any further testimony about
         aggregate doses, it would reflect updated information
9   on all of these points.

10 (Hattis Affidavit at p. 6, Paragraph 17). As is evident, Hattis

11 proposes to make up for these shortcomings at a later time.

12   Plaintiffs do not respond to defendants' pointed criticisms

13 of Hattis' reliance on the Native American fish survey and the

14 1970 U.S. Fish and Wildlife Survey. Rather, they once again

15 attempt to shift attention to the purported failings of HEDR:

16 "Under defendants' view, HEDR's 'maximum' representative

17 individual is so 'hypothetical' so as not to approximate any real

18 user of the Columbia River, in which case the taxpayers have

19 every reason to demand an explanation of how their $27 million

20 was spent for the dose reconstruction." (Plaintiffs' Response

21 Br. at p. 71).

22   Plaintiffs argue Hattis' conclusion that 1 to 5% of his

23 exposed population received doses equaling or exceeding those

24 reported for HEDR's "maximum representative individual" is

25 "consistent" with HEDR's statement that characteristics of

26 "representative individuals" are intended to "approximate those

27

28 **ORDER RE SUMMARY JUDGMENT-   351**

1 of selected segments of the general population." (HEDR River

2 Rpt. at p. 3.25). Once again, the fundamental problem is HEDR

3 did not measure **actual** exposures to the Columbia River population

4 as a whole, to any "selected segment" thereof, or to any "known"

5 individual. Therefore, it is impossible to say the HEDR River

6 Report supports a conclusion that 1 to 5% of the actual exposed

7 population received a dose equaling or exceeding that reported by

8 HEDR for its "maximum representative individual."

9

10 **(d)   Lognormal Distribution**

11 Hattis' median-to-mean conversion formula can be used only

12 where the values (the doses) to be converted are part of a

13 lognormal distribution. Hattis assumes the doses received by his

14 exposed population from the Columbia River were distributed in a

15 lognormal manner.

16 Defendants contend this assumption suffers from two

17 deficiencies. First, because Hattis does not know the doses

18 actually received in any river segment and has no information

19 about actual water and fish consumption practices along the

20 river, defendants say he cannot make any assumption about the

21 distribution of doses attributable to the river. According to

22 defendants, the truth of this is borne out by deposition

23 testimony from Hattis in which he acknowledged that information

24 about actual fish consumption practices along the Columbia River

25 would allow him to test his assumption that the doses have a

26 lognormal distribution. Says Hattis:

27 [I]f there were data on the actual distribution

28 **ORDER RE SUMMARY JUDGMENT-   352**

of fish consumption in the relevant population,
I would surely factor those into the analysis.

I would probably analyze them with a lognormal
distribution because **it's likely they'll turn
out that way** [,] but I would certainly subject
that assumption to a test with the relevant data.

(Hattis Dep. at pp. 141)(Emphasis added).

The second problem, according to defendants, is the HEDR

dose estimates (which Hattis uses for extrapolation purposes) are

not part of the same distribution and thus, cannot be treated as

lognormal. As noted, the "maximum representative individual" is

a person who consumes a substantial quantity of resident fish,

from whence comes the overwhelming majority of his/her radiation

dose.[261] On the other hand, the "typical representative

individual" consumes no resident fish and receives substantially

all of his radiation dose from drinking river water.[262]

Defendants note Hattis' deposition testimony that if he had data

on how many people ate fish and how many did not eat fish within

a given year, he would "try" to analyze the groups separately "if

it were feasible."   (Hattis Dep. at p. 145).

When Hattis plotted the data from the Columbia Intertribal

Fish Commission Study, he dropped the non-fish eaters (10%) and

considered only those who reported eating fish (90%).   (See

Figure 4 at p. 12 of Hattis 1996 Rpt.).   According to Hattis,

---

[261]   According to Table 4.4 of the River Report at p. 4.14,
the "maximum representative individual" at Segment 3 (Pasco)
received 88.5% of his radiation dose from resident fish and
waterfowl while at Segment 12, the figure is 95.3%.

[262]   82.6% at Segment 3; 56.7% at Segment 12.   (HEDR River
Rpt., Table 4.4 at p. 4.13).

ORDER RE SUMMARY JUDGMENT-   353

1 "the lognormal fit is not as good if the non-eaters are

2 included." (Hattis 1996 Rpt. at p. 11).   However, that did not

3 stop Hattis from including in a single lognormal distribution all

4 the non-fish eaters of his entire exposed population.[263]

5   Plaintiffs contend a lognormal distribution is justified

6 because of lognormal distributions for fish-related exposures in

7 three studies cited by Hattis in his 1996 report.   (Hattis 1996

8 Rpt. at pp. 8-10).   One study deals with polychlorinated

9 biphenyl (PCB) levels in the blood of workers in Southern

10 California.   Another study deals with methyl mercury levels in

11 the blood of persons residing in South Haven, Michigan.   Yet

12 another study deals with mercury levels in the blood of Chippewa

13 Indians from Wisconsin.[264]

14 _____

15   [263] In his October 1997 affidavit, Hattis states he "assumed
    that a lognormal distribution would describe the exposures in
16 this population [the entire exposed population], but in principle
    the same technique could be used for subsets of the population,
17 **such as the fraction who actually eat fish from the Columbia
    River."** (Affidavit at p. 1, Paragraph 3) (Emphasis added).   This
18 is an additional concession that fish eaters and non-fish eaters
    should be separated for the purpose of doing a lognormal
19 distribution.

20   [264] In his affidavit, Hattis refers to another study, Rupp,
    E.M., et al., "Some Results of Recent Surveys of Fish and
21 Shellfish Consumption by Age and Region of U.S. Residents," 39
    Health Physics 165-75 (1980), involving a distribution of fish
22 consumption in a national sample of over 24,000 people for the
    years 1973-74.   Hattis says the results of that study "broadly
23 support the use of a log normal distribution" in his risk
    analysis for the exposed Columbia River population.   However,
24 defendants note that Rupp's observations were "neither normally
    nor log normally distributed, but are skewed to the right."
25 (Defendants' Ex. 214 at p. 170).
         On a procedural note, the court fails to see how Hattis can
26 justify resorting to this study in his affidavit when it was
    obviously available well before his original report was written
27 and also before his deposition in September 1996.

28 **ORDER RE SUMMARY JUDGMENT-   354**

1   Defendants correctly point out that these studies have

2   nothing to do with the distribution of fish consumption among

3   users or the Columbia River. They note once again Hattis'

4   statement that it would be helpful to have data on actual

5   distribution of fish consumption in the relevant population (the

6   Columbia River population) so he could "test" his assumption of a

7   lognormal distribution.[265]   (Hattis Dep. at p. 141).

8   All things considered, the court agrees with defendants that

9   Hattis' lognormal distribution is not scientifically reliable

10   because he reasoned from an end result (a log normal distribution

11   of doses) to hypothesize what needed to be known (actual

12   consumption data), but was not known.

13

14   **(e)   Assumption about Water Consumption**

15   More than 80 percent of Hattis' exposed population (487,000

16   residents) is found in Segment 12 (Lower River) which, according

17   to the HEDR analysis, is the lowest river dose location.[266]

18   Segment 1 (Ringold), the maximum dose location, accounts for

19   1,280 residents or 0.21% of Hattis' exposed population.

20   Defendants argue nothing supports Hattis' assumption that all

21   residents of Segment 12 received a radiation dose from the

22   Columbia River. Including them within his exposed population

23   _____

     [265]   Section 2.1.2 of the Hattis Report is titled "Reasons
24   for **Expecting** the Population Distribution of Individual Doses to
     be Lognormal." (Hattis 1996 Rpt. at p. 6)(Emphasis added).

25
     [266]   Defendants suggest all 487,000 residents live in
26   Portland, but as plaintiffs point out, this figure is for all the
     residents of Segment 12 during the relevant time period (1950-
27   1971), including Vancouver, Washington.

28   **ORDER RE SUMMARY JUDGMENT-   355**

1   evidences a result-oriented bias, say defendants.   Defendants

2   rely on their expert, Dr. Frazier.   (Frazier 1996 Rpt. at p. 5).

3   Frazier states the largest contributor to HEDR's dose for

4   the "typical representative individual" is from drinking water

5   (approximately 83 percent of the total dose).[267]   According to

6   Frazier, Hattis assumes this dose typifies the dose received by

7   individuals residing in Segment 12, which includes the residents

8   of Portland, Oregon.   This assumption is significant, says

9   Frazier, because it results in two-thirds of Hattis' collective

10 dose coming from Segment 12.   However, the problem according to

11 Frazier is that Hattis impliedly and errantly assumes Portland's

12 population drank water from the Columbia River.   Frazier notes

13 that in Hattis' analysis of hexavalent chromium (Hattis 1996 Rpt.

14 at p. 27), he (Hattis) found the only cities using the Columbia

15 River as the source of their drinking water were Richland, Pasco,

16 Kennewick and Boardman, Oregon.   These cities have a combined

17 population of 61,940, or less than 11 percent of the total

18 exposed population of 596,000.

19   Plaintiffs argue Hattis had no choice but to include Segment

20 12 in his analysis since HEDR included it in its analysis.

21 According to plaintiffs, if Hattis excluded Segment 12, the

22 defendants would accuse him of omitting the lowest dose location

23 and selecting only segments with higher doses.

24   Plaintiffs assert defendants are in error in stating that

25 _____

26   [267]   The "maximum representative individual" is also assumed
    to have drank river water, but the majority of his radiation dose
27 is assumed to have come from the consumption of fish.

28 **ORDER RE SUMMARY JUDGMENT-   356**

the "typical representative individual" **in Segment 12** received

83% of his/her radiation dose from the Columbia River. They note

that Table 4.4 of the HEDR River Report ("Pathways and

Radionuclides Contributing to Dose, 1956-65") indicates drinking

water made up 56.7% of the total dose for Segment 12 (Lower

River). (River Rpt. at p. 4.13). Table 4.4 indicates for the

"typical representative individual" at Pasco (**Segment 3**),

drinking water made up 82.6% of the total dose.

Plaintiffs note that Table 4.4 shows salmon (a non-resident

fish) made up 2.7% of the total dose for the "typical

representative individual" in Segment 12, while shellfish (a non-

resident fish) made up 40.1%. Plaintiffs contend shellfish and

salmon would "probably" dominate the percentage contribution had

HEDR "included the Phosphorous-32 [P-32] contribution." Table

4.4 shows that P-32 was considered, although perhaps the

plaintiffs are alluding to their argument that HEDR should have

used a higher bioconcentration factor of P-32.[268] That argument

is discussed <u>infra</u>. Nonetheless, if plaintiffs' upward

adjustment for the bioconcentration factor is not justified,

drinking water consumption still makes up the majority (56.7%) of

the "typical representative individual" dose in Segment 12.

According to plaintiffs, Batelle Laboratories (which

performed the HEDR study) explicitly assumed Vancouver,

Washington residents drank water from the Columbia River. They

---

[268] The radionuclides considered by HEDR include: Na-24 (Sodium), P-32 (Phosphorous), Zn-65 (Zinc), As-76 (Arsenic) and Np-239 (Neptunium).

ORDER RE SUMMARY JUDGMENT-   357

1 cite a March 1993 paper by B.A. Napier, one of the authors of

2 HEDR's July 1994 River Report, in which "drinking water

3 transmission factors" were calculated for the purpose of

4 determining the dose received by a "maximum individual" in

5 Vancouver for 1961. B.A. Napier, "Determination of Key

6 Radionuclides And Parameters Related to Dose From the Columbia

7 River Pathway," (March 1993), Appendix C, C.18 (Foulds Ex. 228).

8 In their reply, defendants do not dispute that Portland is not

9 the only community located within Segment 12, that Vancouver is

10 part of Segment 12, and that Vancouver may have received drinking

11 water from the Columbia River. If all of this is correct

12 information, it means some of the 487,000 residents of Segment 12

13 actually did receive drinking water from the Columbia River,

14 contrary to Dr. Frazier's conclusion.[269]

15   Plaintiffs argue that "[b]ecause of the inherently wide

16 variabilities that can influence an individual's radiation dose

17 from multiple pathway river exposure, neither Battelle's, and

18 therefore, Dr. Hattis' assumption [] that 'representative'

19 individuals drank Columbia River water is unscientific where it

20 is part of a multiple pathway analysis." According to

21 plaintiffs, Hattis' acceptance of Batelle's assumption that

22 "representative" individuals across all river segments took

23 drinking water from the river is not inconsistent with his

24 hexavalent chromium analysis in which he identified only four

25 _____

26   [269] Dr. Frazier apparently assumed all 487,000 residents
     were from Portland which did not receive its drinking water from
27   the Columbia River.

28 **ORDER RE SUMMARY JUDGMENT-   358**

1 cities as taking drinking water from the river.

2   Plaintiffs contend the fact Hattis received confirmation as

3 to the four communities (Richland, Pasco, Kennewick and Boardman,

4 Oregon) does not establish that all other communities did **not**

5 receive their drinking water from the river. Plaintiffs say

6 Batelle did not bother to confirm whether or not communities took

7 drinking water from the river, perhaps because records for the

8 time period at issue (1950 to 1971) were neither centralized or

9 complete. Indeed, the only basis on which defendants assert

10 Portland, Oregon did not use the river as a source of drinking

11 water is due to Hattis' failure to include it on his list of

12 communities for which he had confirmed use of the river as a

13 source of drinking water. Plaintiffs note that for his

14 hexavalent chromium analysis, Hattis considered only the drinking

15 water pathway, whereas the radionuclide analysis considers

16 multiple river pathways other than just drinking water (fish

17 consumption, recreation on the water, etc.).

18   Plaintiffs' argument is essentially this: Hattis properly

19 included Segment 12 in his calculation of a population dose

20 because HEDR itself calculated a "typical" and "maximum"

21 representative dose for Segment 12; at least some residents of

22 Segment 12 drank river water; and even if they did not drink

23 river water, they could have received radiation from any number

24 of other sources, including eating fish such as salmon and

25 shellfish.

26   Even assuming Vancouver, Washington residents used the

27 Columbia River as a source of drinking water, that still leaves a

28 **ORDER RE SUMMARY JUDGMENT-   359**

1 majority of Segment 12 residents who did not drink river water.

2 This is because no one can reasonably dispute that the majority

3 of Segment 12 residents are found in Portland, Oregon. According

4 to HEDR, its hypothetical "typical representative individual"

5 still received over half (56.7%) of his radionuclide dose from

6 drinking river water. On top of that, one cannot ignore the

7 other 11 segments. Hattis assumed residents of those segments

8 used the river as a source of drinking water. It appears river

9 water consumption increases as a percentage of total dose the

10 nearer a particular segment is located to the Hanford facility.

11 HEDR states that for Segment 3 (Pasco), river water consumption

12 constitutes 82.6% of the total dose. Therefore, for Segments 4

13 through 11, the percentage is probably less than 82.6%, but

14 probably also **higher** than the 56.7% for Segment 12. Segments 1

15 (Ringold) and 2 (Richland) may even have percentages higher than

16 82.6%.[270] Thus, looking at the segments as a whole, the court

17 must agree that Hattis' population dose is overstated.

18   At his deposition, Hattis acknowledged a substantial portion

19 of the dose received by HEDR's "typical representative

20 individual" was due to drinking water from the river. Hattis

21 said he did not adjust his calculations to account for his

22 information that only four communities had used river water for

23 drinking purposes because "whereas [HEDR] made that clearly

24 ───────────────

25 [270] The HEDR River Report only provides a percentage
breakdown of dose for Segments 3 and 12. The 1956-65 time period
26 is presented because, according to HEDR, it is the period of
highest dose for all locations and all representative individual
27 types.

28 **ORDER RE SUMMARY JUDGMENT-   360**

1 incorrect assumption with respect to drinking water, . . . they

2 also assumed no consumption at all of the fish [resident fish]

3 and . . . that was incorrect in the other direction." (Hattis

4 Dep. at pp. 169-70).[271]

5   Hattis reiterates this in his affidavit in which he claims

6 it is not "consistent" to calculate doses for "representative"

7 individuals who resided in river segments where there was no

8 actual "domestic" use of river water. According to Hattis, this

9 was a "problem" in the HEDR analysis which he was not able to

10 "fix" in the available time. Nonetheless, Hattis thought it

11 "likely that some 'representative' individuals were likely to

12 consume Columbia River fish in amounts delivering doses similar

13 to those estimated via the river pathway by the HEDR

14 investigators." (Hattis Affidavit at p. 2, Paragraph 5)

15   Of course, Hattis' contention would hold more weight if he

16 had data showing **actual** resident fish consumption for his exposed

17 population as a whole. As noted above, the Native American fish

18 survey and the U.S. Fish and Wildlife Survey simply are not

19 adequate for assessing fish consumption for Hattis' entire

20 exposed population. On the other hand, Hattis had actual

21 information that only four communities used the Columbia River as

22 a source of drinking water, which makes it much more difficult to

23 justify an assumption that everyone in his exposed population

24 received radiation from drinking river water. Plaintiffs argue

25 _____

26   [271] The assumption for HEDR's "typical representative
   individual" is he consumed no **resident** fish (as opposed to non-

27 resident or anadromous fish).

28 **ORDER RE SUMMARY JUDGMENT-**   **361**

1 there is uncertainty about how many communities actually used the

2 river as a source of drinking water. However, it is still their

3 burden to come up with the information necessary to support an

4 assumption that all members of the exposed population actually

5 received radiation from drinking river water.

6

7 **(f)   Contrary Data Re Assumptions About Fish Consumption**

8 At his deposition, Hattis confirmed that his research on

9 fish consumption practices, **specifically** on the Columbia River,

10 was essentially confined to the Columbia Intertribal Fish

11 Commission Study. (Hattis Dep. at pp. 95, 132-33). The other

12 survey Hattis relied upon was the U.S. Fish and Wildlife Survey

13 which did not focus specifically on the Columbia River. The

14 limitations of the Native American fish survey and the U.S. Fish

15 and Wildlife Survey have been discussed above.

16 Defendants claim Hattis ignored a number of studies and

17 surveys relating to fishing practices and fish consumption along

18 the Columbia River. According to defendants, these studies

19 "contradict [Hattis'] dose-inflating assumptions about fish

20 consumption" because they show: 1) that less fish were caught

21 along the Columbia River then Hattis assumes were eaten, and 2)

22 Hattis' estimates of fish consumption are overstated by large

23 factors. At his deposition, Hattis stated he was not aware of

24 the studies and had not reviewed them. (Hattis Dep. at pp. 146-

25 53).

26 Hattis testified that based on his assumption that 5 percent

27 of his exposed population had the same dose that HEDR reported

28 **ORDER RE SUMMARY JUDGMENT-   362**

1 for its "maximum representative individual" (an individual who

2 consumed approximately 40 kg of fish), the average **mean** dose for

3 the population in Segment 2 (Richland) would be 453 rem. (See

4 Table 3 of Hattis 1996 Rpt. at p. 14). Hattis indicated that

5 this mean dose corresponded to an annual fish consumption of

6 approximately 12 kilograms (12,000 grams) per person for the

7 entire population of Segment 2. (Hattis Dep. at pp. 113-14).

8 Based on an assumed average population of 25,400 for Richland for

9 the period 1950-1971, Hattis acknowledged the annual fish

10 consumption for the entire Richland population would be "25,400

11 times 12" or 304,800 kilograms. (Id. at pp. 123-25).

12   Defendants refer to a survey conducted by the Washington

13 State Game Department in the 1960s. 1400 fishermen who fished

14 the Columbia River near the Tri-Cities were surveyed. The survey

15 found the annual catch of edible fish from the Columbia River for

16 **all of the Tri-Cities** (Pasco and Kennewick, in addition to

17 Richland) was 10,400 kilograms, significantly lower than 304,800

18 kilograms.[272] Defendants claim that in addition to this survey,

19 two other surveys- a dietary survey of 5,500 students in the Tri-

20

21

22 _____

23   [272] The survey results are found in: 1) Honstead, J.F., et
al., "A Statistical Study of the Habits of Local Fisherman and
Its Application to Evaluation of Environmental Dose Report to the
24 Environmental Protection Agency," (Batelle Research Report for
the Environmental Protection Agency, Y-80054, October 1971); and
25 2) Soldat, J.K., "A Statistical Study of the Habits of Fishermen
Utilizing the Columbia River Below Hanford," **Environmental**
26 **Surveillance in the Vicinity of Nuclear Facilities: Proceedings
of a Symposium Sponsored by the Health Physics Society, January**
27 **24-26, 1968** (1970). (Defendants' Exs. 146 and 144).

28 **ORDER RE SUMMARY JUDGMENT-   363**

1 Cities area schools[273] and a dietary survey of 7,000 adult

2 Hanford employees[274]- show that Richland residents eat on

3 average, less than 500 grams of Columbia River fish annually. Of

4 course, that is significantly less than the 12,000 grams produced

5 by Hattis' analysis.

6   Plaintiffs' assert "<u>Daubert</u> does not entitle defendants to

7 dictate which scientific references are to be relied upon by any

8 expert, particularly since Dr. Hattis relied upon a **credible**

9 **national survey** conducted by a U.S. government agency."

10 Plaintiffs argue Hattis was "under no scientific obligation to

11 adopt incomplete local survey data over national survey data."

12 Plaintiffs assert HEDR rejected the local survey data in favor of

13 surveys conducted by the U.S. Department of Agriculture.

14 Plaintiffs contend the dietary survey of the elementary school

15 children was characterized as "suspect" by HEDR. They also point

16 out what they claim are flaws and limitations of the fishermen

17 survey conducted by the state game department. (Plaintiffs'

18 Response Br. at pp. 72-76).

19   It is not necessary for the court to assess the validity of

20 _____

[273] Soldat, J.K and Honstead, J.F., "Dietary Levels for Tri-
21 City Elementary School Children" (BNWL-CC-1565) (Feb. 26, 1968);
2) Honstead, J.F., "Quantitative Evaluation of Environmental
22 Factors Affecting Population Exposure Near Hanford" (BNWL-SA-
3203) (October 26, 1970); and 3) Endres, G.W.R., et al., "Dietary
23 and Body Burden Data and Dose Estimates for Local School Children
and Teenagers" (Batelle Research Report for the Environmental
24 Protection Agency Y-80054-3)(September 1972). (Defendants' Exs.
147, 148 and 149).
25
[274] Honstead, J.F., "Radionuclide Burden-Diet Relationships
26 Near a Nuclear Facility," **Diagnosis and Treatment of Deposited**
Radionuclides: Proceedings of a Symposium Held at Richland,
27 **Washington 15-17 May 1967** (1968). (Defendants' Ex. 151).

28 **ORDER RE SUMMARY JUDGMENT-   364**

1   the local survey data and whether Hattis should have employed it

2   in his population risk analysis.[275] This is because, as

3   discussed above, the **data which Hattis did use**- the Native

4   American fish survey and the U.S. Fish and Wildlife Survey (which

5   plaintiffs refer to as their "credible national survey")- does

6   not support his assumption that 1 percent or 5 percent of the

7   population at each river segment had the same dose as HEDR's

8   "maximum representative individual." From that assumption,

9   Hattis derives his average mean doses for each segment.

10   Accordingly, the data upon which Hattis relies does not

11   reasonably support his conclusions about the exposure of his

12   Columbia River population **as a whole**. It does not support his

13   average mean dose for Richland of 453 rem and the 12,000 kilogram

14   annual consumption of fish that goes along with it.[276]

15   With regard to Hattis' deposition testimony that his mean

16   dose (453 rem) corresponded to an annual fish consumption of

17   approximately 12 kilograms (12,000 grams) per person for the

18   entire population of Segment 2, plaintiffs say this is a "rough

19   estimate made by Dr. Hattis at defense counsel's request, which

20   _____

21   [275] Defendants dispute plaintiffs' contention that HEDR
     rejected the local survey data. Although HEDR may have used
22   national consumption data for the purpose of gauging **food**
     consumption, defendants say it is clear local **fish** consumption
23   data was used to hypothesize about fish consumption of the
     "typical" and "maximum representative individual." Defendants'
24   argument that HEDR relied on the local data from the Batelle
     surveys appears valid.

25
         [276] As noted above, the extent of exposure depends in
26   significant part on fish consumption since that is how HEDR's
     "maximum representative individual" receives the majority of his
27   radiation dose.

28   **ORDER RE SUMMARY JUDGMENT-   365**

1 analysis ignores the contribution to dose from waterfowl, salmon,

2 drinking water, immersion, etc." They assert that "[a]ny attempt

3 to correlate the dose within Dr. Hattis' lognormal distribution

4 to resident fish consumption must include the contributions from

5 the other pathways."

6   Whether or not a "rough estimate," Hattis testified that an

7 average mean dose of 453 rem translates into an annual fish

8 consumption of "about 12 kilograms . . . [a]ssuming the same mix

9 of fish and waterfowl everything else . . . ." (Hattis Dep. at

10 pp. 114-15). HEDR's "maximum representative individual," is

11 assumed to have consumed 20 kilograms of waterfowl, in addition

12 to his/her consumption of fish. HEDR lumps resident fish and

13 waterfowl consumption together for the purpose of figuring the

14 percentage to which each pathway contributed to the total dose

15 received by the "maximum representative individual." Thus, for

16 the "maximum representative individual" at Pasco (Segment 3),

17 resident fish and waterfowl consumption constitute 88.5% of the

18 total radionuclide dose, whereas at the Lower River (Segment 12),

19 it constitutes 95.3% of total dose. The other pathways- drinking

20 water, external (i.e. immersion), salmon, and shellfish

21 constitute very small percentages of the total dose. (HEDR River

22 Report, Table 4.4 at p. 4.14).

23   In his 1996 report, Hattis did not emphasize waterfowl

24 consumption. Rather, it is apparent from his report that he

25 considered fish consumption, with good reason, to be the most

26 important component of dose. According to Hattis, "the

27 distribution of fish consumption and fish related exposures is a

28 **ORDER RE SUMMARY JUDGMENT-   366**

key factor in assessing population mean dosage and resulting risks." (Hattis 1996 Rpt. at p. 5). It is for this reason he cited the Native American fish survey and the U.S. Fish and Wildlife Survey.

All things considered, plaintiffs have not provided a compelling reason to ignore the fish consumption figure for Segment 2 agreed to by Hattis at his deposition.

(g) Alleged Deficiencies in HEDR River Report

The majority of plaintiffs' 91 page response brief is devoted to attacking the HEDR River Report and what plaintiffs refer to as its "dose-minimizing" assumptions, including "fish bioconcentration factors, holdup time, and a seasonably disproportionate consumption of fish." Essentially, plaintiffs spend the majority of their brief attempting to defend Hattis' population risk analysis not based on what Hattis did, but on what HEDR allegedly did not do.

As is obvious, Hattis used HEDR to calculate his population dose. The only change Hattis made (in his 1996 report) was to increase his population dose because of his belief that HEDR should have used mean, not median bioconcentration factors in analyzing its "maximum representative individual." (Hattis Dep. at pp. 159-60; Hattis 1996 Rpt. at pp. 3 and 23). Hattis apparently felt this upward adjustment was appropriate considering the significant contribution of resident fish/waterfowl consumption to the total dose received by the "maximum representative individual." (1.98 for Segments 1-6;

ORDER RE SUMMARY JUDGMENT-   367

1.64 for Segments 7-12). (Hattis Rpt. at p. 23). In his 1996

report, Hattis did not criticize HEDR on any other basis. He

testified it was "the only aspect that I felt that I could

analyze at the time." (Hattis Dep. at p. 159).

Plaintiffs criticize the HEDR River Report on a number of

grounds which are **not** addressed in Hattis' 1996 report or any

plaintiffs' expert report. In general, these criticisms include:

1) instead of relying on recommended or "expected"

bioconcentration values[277] reported in the scientific

literature, Battelle chose to reconstruct values based on

historical measurements for which the "critical analytical

procedures . . . are missing and may not have been written"[278];

2) Battelle ignored its auditing and quality control obligation

with respect to reconstruction of river and air pathway doses;

and 3) Battelle relied on data generated by U.S. Testing to

reconstruct bioconcentration factors, although it terminated its

contract with U.S. Testing because of concern about quality

assurance and control.

None of these issues are pertinent to whether Hattis'

_____

[277] A "bioconcentration value" or factor (BCF) represents
the ratio between the concentration of a radionuclide in the
river water and its concentration in an organism, such as a fish.
If the concentration in the river is one part per billion and the
concentration in edible fish muscle is one part per million, the
BCF is 1000. The concentration in the fish is 1000 times greater
than that in the river water.

[278] Plaintiffs say HEDR's reconstructed values are generally
below the values contained in the scientific literature. Based
on the literature, plaintiffs assert a bioconcentration factor of
66,700 should have been used for Phosphorous-32. This is
discussed <u>infra</u> in regard to Hattis' supplemental report.

ORDER RE SUMMARY JUDGMENT-   368

1 population risk analysis "fits" the relevant causation inquiry

2 before the court, or whether his analysis is "scientifically

3 reliable." In his 1996 report, Hattis did not assert the

4 bioconcentration factor should be elevated to reflect values in

5 the scientific literature. He did not advocate the use of new or

6 different data to supply the bioconcentration factor. He merely

7 thought it appropriate, based on the **data used by HEDR (derived**

8 **from historical measurements)**, to use the mean bioconcentration

9 value rather than the median value.[279] Consequently, in

10 attacking the data and the assumptions underlying HEDR,

11 plaintiffs are attacking the very underpinnings of the population

12 risk analysis found in Hattis' 1996 report.

13

14   (h) **Daubert Criteria**

15   The methodological shortcomings in Hattis' population risk

16 analysis become even more glaring when the Daubert criteria are

17 examined. His population risk analysis is not based on matters

18 growing naturally and directly out of research he has conducted

19 independent of this litigation. His opinion was developed for

20 the express purpose of testifying in this litigation. His

21 analysis has not been subjected to normal scientific scrutiny

22 through peer review and publication. These are the two principal

23 ways for showing that evidence satisfies the reliability prong of

24 Daubert. In addition, there is no indication Hattis' methodology

25 is "generally accepted" in the scientific community for purposes

26 ──────────────────

        [279] HEDR's "maximum representative individual" was derived

27 based on historical measurements, not the scientific literature.

28 **ORDER RE SUMMARY JUDGMENT-   369**

1 of causation analysis (as opposed to policy planning for

2 regulatory purposes).

3   For all of the foregoing reasons, exclusion of Hattis'

4 "population risk analysis" is additionally warranted because it

5 is not scientifically reliable.

6

7   **c.   Hattis March 1997 Supplemental Report**

8   In March 1997, Hattis submitted a supplemental report

9 entitled "Implications of Possible Systematic Underestimation of

10 Concentration Ratios for 32P for the Doses Calculated by HEDR for

11 'Maximum Representative Individuals' at Various Locations Along

12 the Columbia River." (Plaintiffs' Appendix 2 re Non-Iodine

13 Claims, Ex. 22). According to Hattis:

> Recently, **new information** has come to light
> that **suggests** that there may be some systematic
> understatements in the historical measurements of
> fish radioactivity levels that were used in
> deriving the concentration ratios for 32P for
> different types of fish. Specifically, laboratory
> records **suggest** that in some cases measurements
> were made several weeks after sample collection,
> but **it is not clear** that the results were adjusted
> for the radioactive decay of several fold that would
> have occurred during that period. Official directions
> for conducting the measurements **appear** to call for no
> decay correction factor to be applied on the assumption
> that analyses would be made within 4 hours of sample
> collection.
>
> Because the affected measurements were directly used
> in the calculation of 32P concentration ratios and
> expected doses for the 'maximum representative
> individuals' modeled in the HEDR program, there are
> direct implications for HEDR's principle dosimetric
> conclusions **which were then the basis for my own**

28 **ORDER RE SUMMARY JUDGMENT-   370**

1   **calculations.**[280]   This report addresses the changes
     that would be needed to correct the HEDR-calculated
2   radiation doses for 'maximum representative
     individuals' at various river segments **if** the 32P
3   concentration ratios were to have been systematically
     underestimated by various amounts.

4
     (Hattis 1997 Rpt. at p. 1)(Emphasis added).

5
         Hattis offers calculations for three "possible" cases in

6
     which 32P concentration ratios may have been underestimated: 1)

7
     an increase of 4-fold in the arithmetic mean fish concentration

8
     ratios (muscle concentration and water concentration) over that

9
     calculated in his prior report (population risk analysis); 2) an

10
     increase to 66,700 in the concentration ratio (BCF) for omnivore

11
     fish; and 3) an increase to 66,700 concentration ratio (BCF) for

12
     all fish.   Based on certain scientific literature[281], Hattis

13
     considered 66,700 the "upper end of the possible concentration

14
     ratios for 32P . . . ."   (_Id_. at p. 2).   Hattis added:

15
                 [T]he latter two possibilities, while not
16   incompatible with the literature values for 32P
                 bioconcentration, **could only be reconciled with**
17   **the HEDR measurements by postulating some systematic**
                 distortion of the data beyond even the long term
18   **consistent neglect of basic procedure for correcting**
                 for delay that would be needed to produce the fourfold
19   **understatement presented by the first scenario.**

20   (_Id_).   (Emphasis added).

21   Hattis presents three tables showing the results of his

22   calculations for each of the three scenarios.   According to

23   _____

24   [280]   This confirms that Hattis' population risk analysis is
     based on HEDR without any alteration of the data and assumptions
25   underlying HEDR.

26   [281]   Poston, T.M., and Klopfer, D.C., "A Literature Review of
     the Concentration Ratios of Selected Radionuclides in Freshwater
27   and Marine Fish," Batelle, PNL-5485 (September 1986).

28   **ORDER RE SUMMARY JUDGMENT-**   **371**

Hattis, "[i]t can be seen that the **hypothesized** changes in 32P

fish concentration ratios would be expected to materially change

the estimated dosage for **heavy consumers of local fish.**" (Id. at

pp. 2-6). (Emphasis added). In other words, the doses are

increased.

Hattis presents six additional tables providing what he

believes are the whole body effective dose equivalents (Rem EDE)

and the red bone marrow and lower large intestine doses received

by "maximum representative individuals" at the different river

segments, taking into account each of his three scenarios (Four

Fold Upward Adjustment in Mean BCF; Increase in 32P BCF to 66,700

for Omnivores; Increase in 32P BCF to 66,700 for All Fish). As

defendants point out, Hattis' organ dose tables (Tables EE and

Tables FF) must be wrong because they are identical to the whole

body doses presented in Table BB. (Id. at pp. 7-10).

According to Hattis, his Tables BB, EE, and FF make it clear

"that under **some** circumstances substantial doses **could** have been

received by a **fraction** of the population." He adds that he has

not "as yet[,] gone on to draw implications from these results

for the population aggregate doses and cancer risks estimated in

[his] prior report." He says that revisions to those estimates

will "require [him] to take into account, among other things,

national and local survey data on sport fish consumption." (Id.

at pp. 8 and 10).

Several things are immediately clear from Hattis'

supplemental report. The first is that Hattis concedes **actual**

consumption data has "implications" for his population doses. Of

ORDER RE SUMMARY JUDGMENT-   372

course, this is what defendants have argued all along: accurate

population doses cannot be calculated based on HEDR's

hypothetical individuals.   It is necessary to have consumption

data pertaining to the actual exposed population.[282]

Secondly, Hattis' supplemental report contains a wholly

different analysis than the population risk analysis found in his

1996 report.   Plaintiffs concede as much, stating that "[s]hould

the Court decide that examination of specific organ doses is

appropriate under general causation **in lieu** of population doses,

Dr. Hattis has provided dose estimates for lower large intestine

(LLI) and red bone marrow (RBM) in his unchallenged report of

March 1997 . . . ."   (Plaintiffs' Response Br. at p. 59)(Emphasis

added).   Plaintiffs say the specific organ doses are based on

"recently discovered documents demonstrating systematic

underestimation of P32 concentrations in fish."   However,

plaintiffs do not say what those specific documents are.

In their response brief, plaintiffs contend defendants have

not challenged Hattis' March 1997 report.   Indeed, defendants'

opening brief, submitted June 1997, restricts itself to pointing

out the deficiencies of Hattis' 1996 report.   It is only in their

reply brief that defendants take on Hattis' March 1997 report.

One of the reasons defendants may have waited is to see if

---

[282]   The census data used by Hattis to determine the average
population along the Columbia River between 1950 and 1971 does
not show the number of persons within the communities who
consumed water or fish from the river, how long they lived in the
chosen communities, how much water or how much fish they
consumed, what kind of fish they consumed, or whether they
received a dose of radiation from the river.

ORDER RE SUMMARY JUDGMENT-   **373**

1 plaintiffs would actually attempt to rely on Hattis' 1997 report.

2 This is most likely the case since defendants argue in their

3 reply brief that Hattis' 1997 supplemental report does not meet

4 the supplementation criteria established by this court.

5   In a March 13, 1996 order, this court laid out strict

6 criteria under which it would allow supplementation of reports:

7 1) the request for supplementation would have to be based on

8 actual documents; 2) there would have to be a compelling

9 demonstration from documents actually produced that it was the

10 only appropriate relief; and 3) the information contained in said

11 documents would have to materially affect prior expert analysis.

12   Based on plaintiffs' representation of a need to research

13 newly available documents concerning the Plutonium Finishing

14 Plant (PFP) at Hanford, this court, in a December 16, 1996 order,

15 granted them additional time to submit supplemental expert

16 reports regarding non-iodine releases. This was conditioned upon

17 the reports meeting the criteria specified above. Plaintiffs

18 were granted until March 3, 1997 to submit supplemental reports.

19   Hattis' March 4, 1997 report is apparently intended by

20 plaintiffs to be one of the non-iodine supplemental reports

21 covered by the court's December 16, 1996 order. Defendants argue

22 it does not meet the supplementation criteria. The court agrees.

23   Defendants contend plaintiffs did not make a "specific

24 request" (in other words, seek leave of the court) to supplement

25 Hattis' 1997 report. Indeed, plaintiffs' response brief appears

26 to assume there is no problem with Hattis' 1997 report and that

27 it is not necessary to provide any justification why the report

28 **ORDER RE SUMMARY JUDGMENT-   374**

1 meets the supplementation criteria. The court's March 1996 order

2 indicates a "specific request" is necessary. However, the

3 court's December 16, 1996 order specifically authorized

4 plaintiffs to submit supplemental non-iodine reports. Therefore,

5 plaintiffs' failure to seek leave of the court for Hattis'

6 supplemental report is not fatal. At the same time, if they

7 thought there was any issue about the propriety of the report,

8 plaintiffs were obliged to handle it in their response brief.

9   In his 1997 supplemental report, Hattis states "new

10 information has come to light," specifically laboratory records

11 suggesting that in some cases measurements were made several

12 weeks after sample collection and possibly not making adjustments

13 for radioactive decay occurring during that period. In a

14 footnote, Hattis cites the "Weekly Environmental Monitoring

15 Analysis" for February 17 and 24, 1967 which, according to

16 defendants, were prepared by General Electric (when GE was the

17 contractor operating the plant). (Hattis 1997 Rpt. at p. 1, n.

18 1).

19   Defendants assert these documents were long available from

20 DOE, but plaintiffs did not request them "until the last minute."

21 Defendants note Hattis was not brought into the case to do a

22 river analysis until March 1, 1996, a mere month before

23 plaintiffs' original non-iodine reports were due (April 1, 1996).

24 (Hattis Dep. at p. 20; Hattis Affidavit at Paragraph 4). There

25 is no allegation from plaintiffs that defendants should have

26 previously turned the documents over pursuant to prior discovery

27

28 **ORDER RE SUMMARY JUDGMENT-   375**

1 requests.[283]

2   In his 1997 report, Hattis appears to imply his "new

3 information" also includes an October 24, 1964 letter from F.E.

4 Holt of General Electric.[284] Hattis says this document shows

5 official directions for conducting the measurements "appeared" to

6 call for no application of a decay correction factor. Defendants

7 claim the letter showed up in plaintiffs' own database of

8 documents as early as November 1995 when the database was

9 produced for the defendants.

10   According to defendants, all of the other documents cited in

11 Hattis' 1997 report were publicly available for years before the

12 April 1, 1996 deadline for original non-iodine reports, and the

13 March 3, 1997 deadline for supplemental non-iodine reports.

14 These documents- listed at footnote 22, pp. 37-38 of defendants'

15 reply brief- include various studies and surveys which were

16 published anywhere from two to twenty five years before the April

17 1, 1996 deadline. Hattis does not explicitly or implicitly claim

18 these documents are part of the "new information" received by

19 him.

20   Plaintiffs make only the barest assertion that "specific

21 organ doses are based on **recently discovered documents**

22 demonstrating systematic underestimation of P-32 concentration in

23 _____

24 [283] Defendants note these documents purport to support only
   Hattis' four-fold upward adjustment in mean fish concentration
   ratios to account for decay factors, but not his increase of the

25 BCF to 66,700.

26 [284] "Sample Sizes Used for Calculating Appendix C Detection
   Limits and Other Pertinent Data" (Letter dated Oct. 26, 1964,

27 HEDR Project Record 4003420).

28 **ORDER RE SUMMARY JUDGMENT-   376**

fish." They do not identify the documents, they do not say how "recently discovered" the documents are, nor why the documents were only "recently discovered." As such, the court could justify striking Hattis' supplemental report on the basis of prong 2 of the supplementation criteria that there be a compelling demonstration from documents actually produced that supplementation is the only appropriate relief.

However, consideration of prong 3- whether the supplemental report materially alters prior expert analysis- leaves no doubt the supplemental report should be stricken. Hattis' supplemental work, by his own admission and the admission of plaintiffs' counsel, is **unrelated** to the population risk analysis contained in his original April 1996 report. Although Hattis raises issues which **might** affect his population doses, no expert has provided a report substantiating any change in the assumptions on which Hattis based his original population risk analysis. Hattis does not change his population risk analysis. He merely suggests some change might be necessary, but expresses no certainty that it will materially alter his prior expert analysis: "I have not, as yet, gone on to draw implications from these results for the population aggregate doses and cancer risk estimated in my prior report."[285]

---

[285] Hattis' 1997 report expresses the possibility of an increase in whole body doses and specific organ doses for HEDR's "maximum representative individual" based on an increase in the BCF. Hattis uses the doses received by HEDR's hypothetical "maximum representative individual" to derive the population dose in his 1996 report. However, Hattis' 1997 report says nothing about the specific consequences for his prior population risk analysis. And obviously, Hattis' population risk analysis did

ORDER RE SUMMARY JUDGMENT-   377

1   The court will strike Hattis' 1997 supplemental report

2 because it does not meet the supplementation criteria. However,

3 even if the report was not stricken, it would be of no value

4 since it does not contain any actual opinions.

5   Hattis' supplemental report contains an analysis different

6 from the population risk analysis contained in his original

7 report. Hattis calculates doses for specific organs; computes

8 individual doses for the "maximum representative individual;" and

9 tries to show what the doses would be **if** there were errors in

10 HEDR and **if** the assumptions underlying HEDR's river dose model

11 were different.[286]

12   The conditional nature of Hattis' supplemental report is

13 manifest from its opening paragraphs: new information which

14 "suggests" systematic understatements in the historical

15 measurements of fish radioactivity levels; laboratory records

16 "suggest" that in some cases measurements were made several weeks

17 after sample collection; "not clear" that results were adjusted

18 for radioactive decay; official direction for conducting

19 measurements "appear" to call for no decay; report addresses

20 changes needed to correct HEDR-calculated radiation doses **"if the**

21 _____

22 not address anything about specific organ doses.

23   [286] In his 1996 report, Hattis did not dispute those
   underlying assumptions which are based on historical data about
24 the river. In his 1997 report, Hattis relies on the scientific
   literature in an attempt to dispute HEDR's underlying assumptions
25 about the bioconcentration factor in fish. This is the argument
   which consumes most of the plaintiffs' brief, but which is not
26 supported by any expert opinion. Plaintiffs do not cite Hattis'
   1997 supplemental report as support for any of their arguments
27 that HEDR erred in relying on historical measurements.

28 **ORDER RE SUMMARY JUDGMENT-   378**

1 **32P concentration ratios were to have been systematically**

2 **underestimated by various amounts;"** latter two possibilities

3 (pertaining to increase in BCF to 66,700) "could only be

4 reconciled with HEDR measurements by **postulating** some systematic

5 distortion of the data. . . ."

6   Hattis does not opine that decay was in fact not accounted

7 for in the historical measurements. As defendants note, he cites

8 only documents pertaining to a one month period in 1967, even

9 though his analysis covers more than forty years (1944-1992).

10 Hattis indicates the 66,700 BCF is the "upper end of the range of

11 **possible** concentration ratios" for P-32, but he does not opine

12 what the BCF should actually be, nor is there an analysis showing

13 the BCFs used by HEDR are in error. All Hattis does is crunch

14 the numbers for three "possible" cases based on unsubstantiated

15 assumptions. Finally, as pointed out above, he does not draw any

16 implications on how all of this affects his previous population

17 risk analysis.

18   Hattis' 1997 supplemental report is a "non-opinion" which

19 does not advance plaintiffs' case in any respect.[287] For this

20 reason as well, the supplemental report will be excluded.

21 //

22 //

23 //

24
_____

25   [287] According to defendants, applying Hattis' new analysis
to his population risk analysis would produce over 53,000 excess

26 cancers, meaning that river exposures would be responsible for
cancer in 1 out of every 11 of Hattis' exposed population

27 (596,000 persons).

28 **ORDER RE SUMMARY JUDGMENT-**   **379**

d.   Hattis' October 1997 Affidavit

Hattis' October 1997 affidavit has already been referred to several times in the discussion of Hattis' 1996 population risk analysis.   In various respects, the affidavit confirms deficiencies in Hattis' population risk analysis (i.e. need for actual fish consumption data).   Even Hattis states "more should be done to refine [his] final estimates of the likely health harm via the river pathway."   (Hattis Affidavit at Paragraph 4, pp. 1-2).

Pages 7-26 of Hattis' affidavit contain twenty tables purporting to show various organ doses for "Maximum Representative Individuals' at Different River Segments for Different Scenarios."   Hattis states these tables "extend and revise [his] supplementary report of March 4, 1997. . . ."   In his supplemental report, Hattis provides doses for red bone marrow and lower large intestine, but as noted, those figures must be wrong since they are the same as the whole body doses reported by him.   According to plaintiffs, Hattis' affidavit provides corrected doses.   The other organ doses provided in the affidavit- adrenal, bladder, bone surface, breast, stomach, small intestine, upper large intestine, kidney, liver, lung, ovary, pancreas, skin, spleen, testes, thymus, thyroid and uterus- are all new.   They are not found in the supplemental report.

An affidavit is not the appropriate means for "revising or extending" any expert report.[288]   Furthermore, the court is

_____

[288]   "Revising" and "extending" amounts to changing the prior reports, rather than clarifying them.

ORDER RE SUMMARY JUDGMENT-   380

1 striking the supplemental expert report which the affidavit

2 purports to "revise and extend." Therefore, the tables contained

3 in the affidavit will also be ignored. Like the tables found in

4 Hattis' supplemental report, the tables in his affidavit-

5 pertaining to individual organ doses- have nothing to do with the

6 population risk analysis contained in his original report. The

7 supplemental report and the affidavit are effectively a

8 concession that population risk analysis does not help plaintiffs

9 meet their causation burden of proof, where that burden is to

10 show a "doubling of the risk" in **any** individual plaintiff.

11

12   **e.   Other Expert Reports**

13   The plaintiffs contend that in addition to Hattis' reports,

14 they have other expert reports pertaining to radioactive

15 exposures from the Columbia River. The experts include: 1) Dr.

16 Kenneth McNeil who plaintiffs say calculated the releases of

17 neptunium-239 (Np-239) and plutonium-239 (Pu-239) resulting from

18 fuel element ruptures in the Hanford reactors; and 2) Mr. Tad

19 Deshler, an aquatic biologist, who plaintiffs say calculated the

20 concentration of Np-239 and Pu-239 resulting from activation of

21 natural uranium in reactor cooling water.

22   Dr. McNeil and Mr. Deshler offer nothing about dose or risk.

23 There is no indication Hattis relied on them for any of his dose

24 and risk analyses. Plaintiffs admit McNeil's release estimates

25 were not included in Hattis' analysis of collective dose.

26 (Plaintiffs' Response Br. at p. 86). At his deposition, Hattis

27 acknowledged he had not relied on McNeil for his (Hattis')

28 **ORDER RE SUMMARY JUDGMENT-   381**

1 calculations of the concentrations of radionuclides in the river.

2 (Hattis Dep. at p. 31). Obviously, if Hattis had relied on

3 either McNeil or Deshler, they would only have gotten as far as

4 he did, which is nowhere.[289]

5   Plaintiffs indicate they are submitting under the cover

6 letter of Robert C. Fadeley, his 1965 article entitled "Oregon

7 Malignancy Pattern Physiographically Related to Hanford

8 Washington Radioisotope Storage," **Journal of Environmental**

9 **Health**, Vol. 27, No. 6, May-June 1965, pp. 883-97. (Foulds Ex.

10 173). Fadeley compiled the incidence of cancer in each Oregon

11 county for a six year period starting in 1959. He found:

12   The malignancy indices for counties
             bordering the Columbia River correlate
13   significantly with a mathematical expression
             of exposure to the river and closeness to
14   . . . Hanford.

15 (Id. at p. 883). Plaintiffs concede, however, that Fadeley could

16 not draw any definitive cause-effect relationship between

17 radioactive contamination of the Columbia River Basin and the

18 incidence of cancer in Oregon.

19   Causation is precisely the issue before the court.

20 Fadeley's article says nothing about dose or risk. Consequently,

21 _____

22   [289] Plaintiffs' counsel suggests there is something
    significant about the reports of McNeil and Deshler because they
23 analyze the release of plutonium to the river, which is not one
    of the specific radionuclides considered in HEDR's River Report.
24 HEDR considered neptunium, sodium, arsenic, zinc and phosphorous.
    According to plaintiffs' counsel, neptunium eventually "converts"
25 to plutonium.
         Even if HEDR's failure to specifically consider the release
26 of Pu-239 is significant, the fact is that without Hattis, there
    is nothing at all analyzing the conceivable risk posed by
27 plutonium or any other radionuclides in the river.

28 **ORDER RE SUMMARY JUDGMENT-**   382

1 it is irrelevant to the generic inquiry before this court: was

2 **any** plaintiff's risk of cancer doubled?

3

4   **f. Conclusion**

5   The court will exclude Hattis' original report, his

6 supplemental report, and his affidavit. The court will also

7 exclude the reports of Deshler, McNeil, and Fadeley.

8   **Assuming** the HEDR Columbia River Pathway Integrated Codes

9 are scientifically reliable (STRRM, WSU-CHARIMA, and CRD

10 (Columbia River Dosimetry)), plaintiffs will be limited to

11 relying upon them for estimating the river component of any

12 radiation dose received by them.

13

14   **C. Non-Iodine Exposures**

15   In addition to radioiodine, Hanford emitted quantities of

16 plutonium-239, ruthenium-106, strontium-90, cesium-137, and

17 cerium-144 to the atmosphere. Plutonium is the primary

18 radionuclide at issue insofar as the non-iodine exposures.[290]

19

20   **1. Health Effects**

21   **a. Non-Cancer Claims**

22   Defendants seek summary judgment and dismissal of all claims

23 based on health effects not specifically covered by plaintiffs'

24 _____

25   [290] The court notes that in this "non-iodine" portion of the
case, plaintiffs' experts have provided information allowing for
26 quantitative risk assessment of **iodine** exposure and certain non-
thyroid cancers, including: 1) breast (female); 2) salivary
27 gland; 3) stomach; and 4) bladder. This is discussed <u>infra</u>.

28 **ORDER RE SUMMARY JUDGMENT-   383**

expert reports, including such things as gray hair and

hemorrhoids. In their response, plaintiffs do not dispute that

the only health effects at issue involve various types of non-

thyroid cancer. Accordingly, the court will grant summary

judgment and dismiss all non-cancer claims resulting from alleged

exposure to non-iodine radiation emissions.[291]

b. Non-Thyroid Cancer Claims

(1) Hodgkin's Disease; Cervical Cancer; Uterine Cancer;
Melanoma Skin Cancer; Chronic Lymphocytic Leukemia

Defendants contend plaintiffs have not adduced evidence that

radiation is even "capable of causing" these particular cancers.

Thus, for Hodgkin's disease, defendants cite Dr. Radford's

deposition testimony that he was "not asserting that Hodgkin's

disease is radiogenic at this point." (Radford Dep. at p. 178).

With regard to cervical and uterine cancer, defendants cite the

Thompson A-Bomb study which did not find an association between

radiation exposure and these conditions. (Thompson, et al., 1994

at pp. S50 and S51). Defendants observe that Radford excluded

melanoma skin cancer from his list of cancer risk co-efficients.

(Radford 1996 Non-Iodine Rpt. at pp. 12 and 17).[292] They also

note that Radford specifically excluded chronic lymphocytic

_____

[291] In the iodine case, the only non-cancer claims which
survive are those based on non-autoimmune hypothyroidism, a non-
neoplastic disease.

[292] The list does include **non-melanoma** skin cancer.
Hereinafter, Radford's non-iodine report shall be referred to as
"Radford Rpt."

ORDER RE SUMMARY JUDGMENT-   **384**

1   leukemia from his leukemia risk co-efficient. (Id. at p. 18).

2   According to the BEIR V committee, "[n]o excess cases of chronic

3   lymphocytic leukemia have been observed." (BEIR V at p. 243).

4   Plaintiffs concede the epidemiological data has not yet

5   revealed excess relative risks (ERRs) for these cancers due to

6   radiation exposure. However, they argue these cancer sites are

7   nonetheless sensitive to radiation. They cite as support the

8   recent Pierce study- D.A. Pierce, et al., "Studies of the

9   Mortality of Atomic Bomb Survivors. Report 12, Part I. Cancer:

10 1950-1990," 146 **Radiation Research** 1-27 (1996) (hereinafter,

11 "Pierce, et al., 1996").[293] According to Pierce:

12   Generally . . . it should be understood
                that a low ERR may not so much indicate
13   that a site is 'less sensitive' to radiation,
                but rather that some factors which contribute
14   to the background rate act more additively
                than multiplicatively with radiation effects.
15
    (Id. at p. 16).
16
         Based on Pierce, plaintiffs assert Hodgkin's disease,
17
    cervical cancer, uterine cancer, melanoma skin cancer, and
18
    chronic lymphocytic leukemia "presumably appear at doses between
19
    2 and 5 rem, or lower, but their impact in epidemiological data
20
    is masked by other predominant causes." According to plaintiffs,
21
    at the individual causation stage, plaintiffs with these cancers
22
    could present testimony from medical experts showing either a
23
    special radiation sensitivity or ruling out, or significantly
24
    downgrading, alternate causes of a cancer (aka "differential
25
    diagnosis").
26
    _____

27   [293] Plaintiffs' Ex. 52 to Appendix 4 re Non-Iodine Claims.

28 **ORDER RE SUMMARY JUDGMENT-   385**

1   Several things need to be pointed out here. First of all,

2   plaintiffs do not cite to any of their expert **reports** as support

3   for the proposition that radiation is "capable of causing" any of

4   these particular cancers. Rather, they cite to scientific

5   literature without any expert interpretation thereof. For

6   example, plaintiffs claim that "in the studies to date, the

7   effects of radiation on cervical cancer are apparently swamped by

8   the predominant causal factor, human papilomavirus (HPV)." They

9   then cite to scientific literature[294] which purportedly

10   establishes that while infection with HPV is necessary for

11   cervical cancer to occur, it is not sufficient by itself. (See

12   footnotes 108-112 at pp. 109-111 of Plaintiffs' Joint Response To

13   Defendants' Motion For Summary Judgment Re Non-Iodine).

14   Even were this enough for these cancer claims to pass into

15   Phase III on a "capable of causing" standard, the plaintiffs

16   would not have any evidence to present to a jury. The plaintiffs

17   need experts to explain to a jury what the scientific literature

18   means.

19   Secondly, the court has determined the relevant evidentiary

20   standard at this stage of the proceedings is whether radiation is

21   a "more likely than not" cause of the cancer. Because cancer can

22   be caused by sources other than radiation, and there is no

23   biologically or pathologically certain way to distinguish a

24   radiation induced cancer from a non-radiation induced cancer,

25   _____

     [294]   Daling, et al., "The Relationship of Human
26   Papilomavirus-Related Cervical Tumors to Cigarette Smoking, etc.,
     Cancer Epidemiology," **Biomarkers and Prevention**, Vol. 5: 541-48
27   (1996), at p. 541.

28   **ORDER RE SUMMARY JUDGMENT-   386**

1 epidemiological proof is necessary for establishing the dose at

2 which it is reasonable to infer radiation exposure is a "more

3 likely than not" cause. For these particular cancers, the

4 plaintiffs have no epidemiological proof.[295] They do not have

5 it now and there is no indication they will have it later.

6   Plaintiffs suggest that by way of medical expert testimony

7 at trial they can prove these particular cancers were radiation

8 induced. This simply is not possible under the applicable "more

9 likely than not" standard. Having a doctor testify an individual

10 is radiation sensitive (if that can actually be established) or

11 "downgrade" potential alternate causes is not sufficient, by

12 itself, to sustain a jury verdict that radiation, and in

13 particular Hanford radiation, is a "more likely than not" cause

14 of a cancer. Existing scientific knowledge does not allow any

15 physician to testify to a reasonable medical certainty that

16 radiation is the cause of an individual's cancer. In the absence

17 of epidemiological proof, he or she cannot even testify in terms

18 of "probabilities." He/she can only discuss "possibilities."

19   Accordingly, the court will grant summary judgment on all

20 plaintiffs' claims for Hodgkin's disease, cervical cancer,

21 uterine cancer, melanoma skin cancer, and chronic lymphocytic

22 leukemia. All such claims will be dismissed with prejudice.

23 //

24 //

25 ───────────────

    [295] Not only does Radford not provide risk co-efficients for

26 melanoma skin cancer and chronic lymphocytic leukemia, he does
not provide them for cervical cancer, uterine cancer, or

27 Hodgkin's disease. (Radford Rpt. at pp. 17-18).

28 **ORDER RE SUMMARY JUDGMENT-   387**

1   **(2)   Prostate Cancer; Non-Hodgkin's Lymphoma; Cancer of**

2   **the Esophagus; Gallbladder Cancer; Cancer of the Oral**

3   **Cavity and Pharynx, Including Salivary Glands; Cancer**

4   **of the Nasal Cavity**

5

6   Radford's risk co-efficient for prostate cancer is 0.29 per

7   Sievert (100 rem), corresponding to a doubling dose of 345,000

8   millirem. (1.0 (100%)/0.29 (29%)= 3.45 Sievert or 345 rem).[296]

9   (Radford Dep. at p. 157). Defendants argue that although Radford

10   provided this risk co-efficient, he was unable to cite any study

11   showing that radiation causes prostate cancer (i.e. is "capable

12   of causing" prostate cancer). They note that Radford

13   acknowledged his risk co-efficient (0.29 per Sievert) was not

14   "statistically significant" from zero. (Id. at p. 157).

15   In his report, Radford indicated the risk co-efficient for

16   non-Hodgkin's lymphoma is 0.30 per Sievert (derived from Preston,

17   et al., 1994)[297], corresponding to a doubling dose of 333,000

18   millirem. (Radford Rpt. at p. 18). He indicated an excess was

19   not found for females. For males, the excess relative risk per

20   Sievert (ERR/Sv) was 0.62 "with a slightly higher value for those

21   exposed below age 20." (Id. at p. 16). At his deposition,

22   Radford testified he arrived at the 0.30 per Sievert figure by

23   —————————————————

24   [296] Defendants use rounded off doubling dose figures in each
case, none of which are disputed by the plaintiffs for any of the
cancer sites discussed herein.

25

26   [297] Preston, et al., "Cancer Incidence in Atomic Bomb
Survivors, Part III: Leukemia, lymphoma, and multiple myeloma
1950-1987," **Radiation Research**, 137: S68-S97 (1994).

27   Plaintiffs' Ex. 44 to Appendix 4 re Non-Iodine Claims.

28   **ORDER RE SUMMARY JUDGMENT-   388**

1 averaging between males (.6) and females (0). (Radford Dep. at

2 p. 180). Radford appears to have reversed course on this at his

3 deposition, testifying he would "prefer to use .6 for males," and

4 "nothing for women." (Id.) The corresponding doubling dose for

5 an ERR/Sv of 0.62 is 167,000 millirem.

6   Defendants apparently contest whether there is sufficient

7 evidence showing radiation is even "capable of causing" non-

8 Hodgkin's lymphoma. They cite UNSCEAR 1994 which reported "there

9 is no convincing evidence that non-Hodgkin's lymphoma is

10 associated with radiation exposure." (UNSCEAR 1994 at p. 33,

11 Defendants' Ex. 121).

12   In his report, Radford reported for cancer of the esophagus

13 an ERR/Sv of 0.28 for "all ages." He derived this figure from

14 the Thompson A-Bomb study. He described this as a "composite"

15 figure, noting that for males the figure was .04 and for females

16 1.83. Radford observed that for females below the age of 20, no

17 cases had been found. (Radford Rpt. at p. 10). At his

18 deposition, Radford testified he would use his "composite" figure

19 of 0.28 "with a little sex difference thrown in." Consequently,

20 he would be inclined to use 0.25 ERR/Sv for males and 0.35 ERR/Sv

21 for females **over the age of 20**. (Radford Dep. at p. 138). This

22 corresponds to respective doubling doses of 400,000 millirem for

23 males and 286,000 millirem for females over age 20. Radford

24 indicated that no significant risk had been observed yet in

25 females ages 20 and under. (Id.)

26   Defendants contest whether there is sufficient evidence

27 showing that radiation is "capable of causing" esophageal cancer.

28 **ORDER RE SUMMARY JUDGMENT-   389**

1   They cite the Thompson study which concluded the results of its

2   analysis "did not establish an overall association between cancer

3   of the esophagus and radiation exposure." (Thompson, et al.,

4   1994 at p. S34).

5   For gallbladder cancer, Radford reported an ERR/Sv of 0.12

6   which corresponds to a doubling dose of 833,000 rem. He

7   indicated the excess was "entirely due to females," with a higher

8   co-efficient for females age 10-19. (Radford Rpt. at p. 11).

9   Nonetheless, at his deposition, Radford testified that using the

10 figure of 0.12 for all ages and both sexes was warranted.

11 (Radford Dep. at p. 146).

12   Defendants contest whether there is sufficient evidence

13 showing that radiation is even "capable of causing" gallbladder

14 cancer. Once again, they cite Thompson which found "no evidence

15 of an association with dose . . ., age at exposure, time since

16 exposure or attained age," and concluded from a review of other

17 studies reporting negative results that "the gallbladder appears

18 relatively insensitive to radiation carcinogenesis." (Thompson,

19 et al., 1994 at p. S41). Plaintiffs do not dispute that Thompson

20 did not find a statistically significant excess of gallbladder

21 cancer among the atomic bomb survivors.

22   For cancer of the oral cavity and pharynx (lip, tongue,

23 etc.), excluding the salivary gland, Radford reported an ERR/Sv

24 of 0.29 for all ages and sexes (Radford Rpt. at p. 9),

25 corresponding to a doubling dose of 345,000 millirem. Defendants

26 note that Radford discusses the "statistical uncertainty" of the

27 Thompson data from which his risk co-efficient is derived:

28 **ORDER RE SUMMARY JUDGMENT-   390**

> For this group of cancers and for all ages,
> the ERR/Sv was 0.29 for both sexes, 0.16 for
> males and 0.46 for females. For both sexes
> the rates were highest for those age 0-9 at
> exposure, less for ages 10-19, and zero over
> the age of 40. For ages 20-39 at exposure [,]
> males showed no excess but females did. Those
> numbers have **substantial statistical uncertainty
> arising from the relatively small number of cases,
> 132 for all ages and both sexes.**

(Radford Rpt. at p. 9)(Emphasis added). In citing this,

defendants apparently suggest Thompson does not allow for an

inference that radiation is even "capable of causing" cancer of

the oral cavity and pharynx.

For salivary gland cancer, Radford reported an ERR/Sv of 3.0

"with perhaps 6 for children and about 10 for infants." (Radford

Rpt. at p. 10). An ERR/Sv of 6.0 corresponds to a doubling dose

of 17,000 millirem. An ERR/Sv of 10.0 corresponds to a doubling

dose of 10,000 millirem. It does not appear defendants raise an

argument about the capacity for radiation to cause cancer of the

salivary gland.

For cancer of the nasal cavity, Radford reports an ERR/Sv of

0.22. (Radford Rpt. at p. 14). This corresponds to a doubling

dose of 455,000 millirem. The defendants point out that the

Thompson data, from which Radford derived his risk co-efficient,

was not statistically significant. According to Radford:

> The exposed subjects had a significantly
> increased risk of nasal cancer, compared with
> the controls. . . . When the data were tested for a
> dose-response relationship, however, there was no
> significant trend, indicating that the relatively
> small number of cases in the exposed group (34) was
> insufficient for this purpose.

(Id.). Apparently, defendants are suggesting Thompson does not

ORDER RE SUMMARY JUDGMENT-   391

1 even allow for an inference that radiation is "capable of

2 causing" cancer of the nasal cavity.

3   Whether radiation is "capable of causing" these various

4 cancers is not the question which ultimately needs to be

5 satisfied in this litigation. Nonetheless, unless the plaintiffs

6 have at least raised an issue of material fact that radiation is

7 "capable of causing" them, Radford's reported risk co-efficients

8 are worthless. The risk co-efficients are used to tell us at

9 what dose levels an inference can be raised that radiation is a

10 "more likely than not" cause of these cancers. If radiation is

11 not "capable of causing" the cancer, it obviously will not

12 qualify as a "more likely than not" cause.

13   Accordingly, the court will first address the issue of

14 whether plaintiffs have presented sufficient evidence to raise an

15 issue of material fact that radiation is "capable of causing"

16 non-Hodgkin's lymphoma, esophageal cancer, gallbladder cancer,

17 prostate cancer, cancer of the nasal cavity, and cancer of the

18 oral cavity and pharynx. Plaintiffs claim this is all they need

19 to do at this phase of the proceedings, but the court has

20 determined otherwise. Therefore, even if the court concludes

21 there is an issue of material fact in this respect, it then needs

22 to determine whether there is any issue of material fact that

23 radiation is a "more likely than not" cause of any of these

24 cancers.

25

26   **(a) "Capable of Causing"**

27   According to plaintiffs, they demonstrate the "generic

28 **ORDER RE SUMMARY JUDGMENT-   392**

1 causal association" between non-iodine exposures from Hanford and

2 their specific cancers by two means: 1) "the undisputed

3 underlying biological basis for radiation-induced cancer

4 induction for the cancers at issue;" and 2) "detailed, human-

5 based, epidemiological evidence showing definitively that

6 radiation **exposures in dose ranges [consistent] with Hanford**

7 **radionuclide exposure are capable of causing, and capable of**

8 **substantially contributing to the causation of plaintiffs'**

9 **cancers."** (Emphasis in text of Plaintiffs' Joint Response Brief

10 at pp. 74-75).

11   As plaintiffs point out, defendants do not appear to dispute

12 the underlying biological basis or "biological plausibility"[298]

13 for radiation induced cancer. However, "biological plausibility"

14 is but one factor considered in determining whether radiation is

15 "capable of causing" a particular cancer. "Strength of

16 association" is another factor, and the one relied upon by

17 defendants. Defendants claim the statistical association between

18 radiation and non-Hodgkin's lymphoma, esophageal cancer,

19 gallbladder cancer, prostate cancer, cancer of the nasal cavity,

20 and cancer of the oral cavity and pharynx, is so **weak** that an

21 inference of generic causal association cannot be drawn,

22 regardless of "biological plausibility." As pointed out

23 previously, there must be a strong enough statistical association

24 and there must be temporality (exposure preceded onset of the

25 _____

26   [298] Plaintiffs readily acknowledge "the mechanisms of cancer
   induction have not been fully worked out." Plaintiffs' Joint
27 Response at p. 77.

28 **ORDER RE SUMMARY JUDGMENT-   393**

1   disease), otherwise "biological plausibility" and all of the

2   other epidemiological criteria are of no avail.

3   Plaintiffs assert "statistical significance" is no longer of

4   consequence in the radiation/cancer causation context. According

5   to plaintiffs, "the evidence of causal association between

6   radiation and cancerous tumors is now so overwhelming that the

7   association is presumed for **all** cancer sites unless there is

8   evidence to the contrary." They base this argument on the Pierce

9   study:

10   First, even if the ERRs were identical, sites
               with fewer numbers of cancer deaths are likely
11   not to show statistical significance due simply
               to lack of statistical power. Second, it is
12   difficult to formulate an adequate test procedure
               when the risks depend on other factors such as
13   sex or age at exposure. Finally, this approach
               is rooted in the notion that one should tentatively
14   conclude that there is no risk at a given site unless
               the data show differently. **With regard to this**
15   **last point, while testing for no effect is an**
               appropriate starting point in most scientific
16   **investigations, in view of the accumulated data**
               on radiation and cancer it would seem appropriate
17   **to consider the null hypothesis of interest as being**
               that <u>site specific ERRs are similar for all solid</u>
18   **<u>cancers</u>.**

19   (Pierce, et al., 1996 at p. 15)(Emphasis added).

20   In other words, say plaintiffs, the lack of "statistical

21   significance" at a particular solid tumor cancer site is not

22   evidence of no effect, but because of the "near universal

23   acceptance" of the causal association between radiation and

24   cancer, an effect should be assumed unless proven otherwise.

25   Radford made this point during his deposition:

26   What they [Pierce, et al.] did was to ask
               the question, do the excesses in all of these
27   various cancers differ significantly from the

28   **ORDER RE SUMMARY JUDGMENT-   394**

average excess for all cancers? And when they
did the calculations, they found that indeed,
none of these cancers differed significantly
from the other cancers.

So the conclusion that one would draw from
this is that radiation is causing cancer in
a wide variety of organs, and they are not
statistically significantly different from
each other.

So that's a very different approach that's
been used in the past, and I can say from
personal experience that often, in the past,
people would say, well, cancer of the prostate
isn't statistically significantly different
from no effect; therefore, it doesn't exist.
And that approach, I found not scientific,
because the effect of radiation in producing
cancer is so well documented now, that to say
just because it is not statistically significant,
doesn't mean it doesn't exist.

(Radford Dep. at pp. 472-73).[299]

It is apparently for this reason, Radford testified he

disagreed with the conclusion of the Thompson study that there is

not an association between esophageal cancer and radiation

exposure (Radford Dep. at p. 139 citing the Pierce study); and

that he disagreed with the UNSCEAR conclusion that there is not

an association between radiation exposure and non-Hodgkin's

lymphoma (Radford Dep. at pp. 178-79). Radford readily

acknowledged the lack of "statistical significance" in the

epidemiological data for both gall bladder and prostate cancer

(Radford Dep. at pp. 146 and 157), but asserted that "in this day

and age, with regard to radiation-induced cancers, that is no

---

[299] The Pierce study was not cited in Radford's non-iodine
report, apparently because it was not yet available for his
consideration.

ORDER RE SUMMARY JUDGMENT-   395

1 longer the issue." (Id. at p. 157).[300]

2 Plaintiffs say Radford has concluded that for cancer sites

3 which do not show a statistically significant excess, the Pierce

4 study justifies using the average risk co-efficient for all solid

5 tumors, which is an ERR/Sv of 0.63. Interestingly, plaintiffs do

6 not provide a citation where Radford's conclusion can be found.

7 It appears the reason for this is that it is found only in

8 Radford's post-deposition declaration.

9 In his November 1997 declaration, Radford says: "[W]here

10 the cancers are so rare that epidemiologic studies cannot provide

11 an accurate expression of radiation-induced risk, I believe that

12 the proper approach is to use the average solid cancer risk for

13 all cancers shown by Pierce, et al. to apply to those rare

14 cancers." (Radford November 1997 Declaration at p. 10, Ex. 5 to

15 Plaintiffs' Appendix 1 re Non-Iodine Claims).

16 Obviously, in his report and his deposition, Radford did not

17 use 0.63 ERR/Sv for prostate cancer (0.29), gallbladder cancer

18 (0.12), nasal cavity cancer (0.22), non-Hodgkin's lymphoma

19 (0.60), esophageal cancer (0.28), and oral cavity and pharynx

20 cancer (0.29). Using 0.63 ERR/Sv would significantly increase

21 the doubling dose for each of these cancer sites, with the

22

23

---

24 [300] Radford explains that although the epidemiological data
does not show a statistically significant difference from zero or

25 no effect, that is not the question pursuant to the Pierce study.
The question is whether the excess found for one kind of cancer

26 differs significantly from the excess found for all solid
cancers. (Radford November 1997 Declaration at p. 5, Ex. 5 to

27 Plaintiffs' Appendix 1 re Non-Iodine Claims).

28 **ORDER RE SUMMARY JUDGMENT-   396**

1 exception of non-Hodgkin's lymphoma.[301]

2 Defendants assert this amounts to an _ex post facto_ revision

3 of the expert record. The court agrees. This is a violation of

4 Fed. R. Civ. P. 26(a)(2)(B) (expert report shall contain a

5 complete statement of all opinions to be expressed and the basis

6 and reasons therefor). Radford will be held to the risk co-

7 efficients set forth in his report and reiterated at his

8 deposition.[302]

9 Nonetheless, this still does not answer the question of

10 whether plaintiffs have provided enough evidence to raise a

11 genuine issue of material fact that radiation is "capable of

12 causing" these particular cancers. As further evidence that it

---

[301] An ERR/Sv of 0.63 results in a doubling dose of 158,000 millirem.

[302] Defendants advance several additional arguments against Radford using the 0.63 ERR/Sv as an "average." There is such a blatant violation of Rule 26, the court need not cite any other reasons for prohibiting Radford's use of the 0.63 ERR/Sv figure. However, two of defendants' arguments are especially compelling.
     Defendants contend use of this "average" figure ignores the fact organs differ in their sensitivity to radiation, as reflected in Radford's own risk co-efficients which vary considerably from organ to organ. They correctly note that Radford has not proposed to use the figure in lieu of the values provided in his report, in particular those which **already exceed** 0.63 ERR/Sv. Defendants assert Radford is simply using the figure as "gap-filler" without any epidemiological support.
     Defendants observe that the risk co-efficients presented by Radford in his report are already "averages" which span age and gender categories. Combining the data in this fashion makes it much less subject to "statistical variation." (Radford 1996 Non-Iodine Rpt. at p. 9). In other words, the results may not be statistically significant within a particular age or gender category, but by "averaging," statistical significance becomes less of a problem. Since Radford has already performed an "averaging" to arrive at the risk co-efficients stated in his report, defendants legitimately ask why any additional "averaging" is necessary to increase the risk co-efficient.

ORDER RE SUMMARY JUDGMENT-   397

1 is so capable, plaintiffs cite studies (Pierce and Lubin[303]) and

2 deposition testimony from their experts Modan and Radford, and

3 defendants' expert Mettler, supporting the proposition there is

4 no threshold below which radiation exposure is incapable of

5 causing cancer in general. As far as the court can discern,

6 defendants do not dispute that point.

7   Plaintiffs also assert they can show, "based upon solid

8 epidemiologic data, that radiation exposures as low as two rems

9 have been demonstrated to be capable of causing solid tumor

10 cancers at a high statistical confidence level." For this, they

11 cite Radford's November 1997 declaration. From a review of the

12 data contained in the Pierce study, Radford concludes that "at

13 doses as low as 2 rems there is a significant excess of solid

14 cancers." (Radford Declaration at p. 8). According to

15 plaintiffs, 2 rems is consistent with their exposures to Hanford

16 emissions and therefore, they have met their burden of showing

17 that Hanford emissions are "capable of causing" all of their

18 solid tumor cancers, including prostate cancer, gallbladder

19 cancer, esophageal cancer, non-Hodgkin's lymphoma, cancer of the

20 nasal cavity, and cancer of the oral cavity and pharynx.

21   The plaintiffs acknowledge Radford did not present his "2

22 rem conclusion" in his non-iodine report since the Pierce study

23 "appeared too late for analysis and inclusion . . . ." What

24 plaintiffs do not point out is that neither did Radford state

25   [303] Jay H. Lubin, et al., **Radon and Lung Cancer Risk: A**
26 **Joint Analysis of 11 Underground Miner Studies**, U.S. Department
of Health and Human Services (1994). Plaintiffs' Ex. 40 to
27 Appendix 4 re Non-Iodine Claims.

28 **ORDER RE SUMMARY JUDGMENT-   398**

1   this conclusion at his deposition when the Pierce study results

2   were clearly **available**. At his deposition, Radford cited Pierce

3   for other reasons, specifically for the proposition that

4   "statistical significance" is no longer of importance in the

5   radiation/cancer causation context. The "2 rem conclusion" is

6   asserted for the first time in Radford's November 1997

7   declaration which was filed after both his report (March 1996)

8   and his deposition (November 1996 and February 1997).[304] Thus,

9   here is yet another violation of Fed. R. Civ. P. 26 which was not

10   remedied at the time of Radford's deposition. As is the

11   situation with the 0.63 ERR/Sv "average" risk co-efficient, the

12   defendants have not had an opportunity to depose Radford about

13   his "2 rem conclusion."[305]

14   Disregarding this evidence- the 0.63 ERR/Sv "average" risk

15   co-efficient and Radford's "2 rem conclusion"- the court

16   nonetheless finds plaintiffs have raised a genuine issue of

17   material fact that radiation exposure is "capable of causing"

18

---

19   [304] The same is true with respect to the conclusion
contained in Radford's declaration that there is a significant

20   excess of acute leukemia in the range of 1.5 to 2 rems. Radford
bases this on Stevens, et al., "Leukemia in Utah and radioactive

21   fallout from the Nevada Test Site.," **JAMA** 264: pp. 585-591
(1990). He needed to do so because the Pierce study applies only

22   to "solid" cancers. Leukemia is not a "solid" cancer.

23   [305] There is still technically a violation of Rule 26 when
an expert comes up with a new theory or conclusion at his

24   deposition. However, the point is that at the deposition, there
is an opportunity to ask the expert about his new theory or

25   conclusion and if need be, conduct additional discovery regarding
the new theory or conclusion, including perhaps a second

26   deposition of the expert. Defendants did not have that
opportunity in this case. Therefore, the violation of Rule 26

27   has not been ameliorated. It is prejudicial.

28   **ORDER RE SUMMARY JUDGMENT-**   **399**

prostate cancer, gallbladder cancer, esophageal cancer, non-Hodgkin's lymphoma, cancer of the nasal cavity, and cancer of the oral cavity and pharynx. At his deposition, Radford did assert that Pierce stood for the proposition that lack of statistical significance in the radiation/cancer causation context does not prohibit drawing an inference that radiation is "capable of causing" a particular cancer. Although defendants and their experts may not necessarily agree with that conclusion, they do not mount a challenge to its scientific propriety (i.e. do not seek its exclusion on <u>Daubert</u> grounds).

Accordingly, based on that evidence, as well as the apparently undisputed fact there is a no-threshold dose response curve for radiation and cancer, and granting the plaintiffs all favorable inferences therefrom, an issue of material fact has been raised that radiation exposure is "capable of causing" these particular cancers.

(b)   "More Likely Than Not"

That radiation may be "capable of causing" these particular cancers is not sufficient to have a jury consider claims based on those cancers. Evidence that radiation is "capable of causing" these cancers does not allow a jury to render a verdict that radiation, and in particular Hanford radiation, is a "more likely than not" cause.[306]

---

[306]   Defendants compare radiation dose estimates from Hanford with background radiation dose estimates. However, their summary judgment motion does not turn on this. At trial, this type of evidence may be relevant to the question of whether **Hanford**

ORDER RE SUMMARY JUDGMENT-   400

Radford has provided risk co-efficients for these cancers from which doubling doses can be derived and in turn from which a jury can potentially conclude Hanford radiation is a "more likely than not" cause of the cancer. However, two qualifications are necessary. First, Radford testified he was not willing to use his non-Hodgkin's lymphoma risk co-efficient for females. As such, a jury has no evidence from which to determine that any female plaintiff's non-Hodgkin's lymphoma was caused by Hanford radiation emissions. If any of the plaintiffs claiming non-Hodgkin's lymphoma are female, their claims cannot go forward and will be dismissed. Likewise, any female plaintiffs who are 20 years old or under, and **currently suffering** from esophageal cancer, will have their claims dismissed. Radford testified his risk co-efficient for females was only for females over age 20.

Otherwise, the doubling doses are as follows: 1) Prostate cancer- 345,000 millirem; 2) Non-Hodgkin's Lymphoma (Males Only)- 167,000 millirem; 3) Gallbladder Cancer- 833,000 millirem; 4) Nasal Cavity Cancer- 455,000 millirem; 5) Esophageal Cancer- 400,000 millirem for males; 286,000 millirem for females ages 21 and over; 6) Cancer of the Oral Cavity and Pharynx- 345,000 millirem; 7) Salivary Gland Cancer- 33,000 millirem for adults; 17,000 millirem for "children" and 10,000 millirem for "infants." Exposure to doses at or below these levels warrants dismissal under the "more likely than not" standard.

These doubling doses are not **decreased** by a factor of five

---

emissions are a cause in fact of an individual's cancer.

ORDER RE SUMMARY JUDGMENT-   401

1   to account for Radford's "individual susceptibility factor." The

2   court has found this factor is not scientifically reliable and

3   excluded it on <u>Daubert</u> grounds. The defendants are willing to

4   have their motion decided on the basis of Radford's risk co-

5   efficients, although they argue the co-efficients overstate the

6   risk. Defendants contend one reason is Radford did not consider

7   a dose rate effectiveness factor (DREF).

8   In the iodine portion of the case, defendants argued the

9   doubling doses for thyroid cancer and hypothyroidism need to be

10   **increased** by .66 to account for the difference between external

11   radiation (gamma rays) and internal radiation (via ingestion or

12   inhalation). This is warranted, according to defendants, because

13   the epidemiological studies from which the doubling doses are

14   derived involve exposures to high dose external radiation (i.e.

15   the Thompson A-Bomb study). Defendants assert a DREF is

16   warranted in the non-iodine case for similar reasons: the atomic

17   bomb study involved acute doses of external radiation delivered

18   at a high dose rate and therefore, does not provide direct

19   information on the effects of protracted, internal, or low-dose

20   rate exposures at issue in this case. The theory is that

21   spreading the dose out over time increases the opportunity for

22   biological and cellular repair. Based on recommendations from

23   the ICRP, BEIR V and the NCRP[307], defendants apparently suggest

24   a DREF of at least 2 is appropriate. This would double the

25

---

26   [307] IRCP 1990, Paragraphs 74, B62 at pp. 18-19, 111-12, Defendants' Ex. 53; BEIR V (1990) at p. 23, Defendants' Ex. 6;

27   NCRP 1993 at pp. 1, 8-9, Defendants' Ex. 89.

28   **ORDER RE SUMMARY JUDGMENT-**   **402**

1   doubling doses reported above (and below).

2   The court did not strike Radford's opinion that external

3   radiation and internally deposited **iodine** are equally effective

4   in causing thyroid cancer. The court found there is an material

5   issue of a fact whether a DREF should apply in that situation.

6   The court is compelled to reach the same conclusion here.

7   At his deposition, Radford acknowledged he had not taken

8   into account a DREF for alpha radiation. Alpha radiation is the

9   type of radiation emitted by plutonium. (Radford 1996 Non-Iodine

10   Rpt. at p. 3).[308] Radford referred to the Lubin study which

11   analyzed the effective dose rate of miners exposed to different

12   concentrations of radon. According to Radford, the Lubin study

13   showed the risk co-efficient for lung cancer was markedly

14   dependent on the exposure rate: "With higher exposure rates, the

15   risk was lower, and with lower exposure rates the risk was

16   higher."[309] Radford opined that based on this, the existence of a

17   DREF would have the effect of **increasing** the risk estimates for

18   low dose rate exposures, "perhaps substantially." (Radford Dep.

19   at pp. 479-80). In other words, Radford suggests that with

20   regard to an alpha emitter like plutonium, it is scientifically

21   appropriate to increase the risk co-efficients (and lessen the

22   doubling doses) for low dose rate exposure, as compared to the

23   risk co-efficients derived from high dose studies.

_____

[308] The court fails to see where Radford discussed DREF in his non-iodine report.

[309] Plaintiffs say the Pierce study has also reported similar results: higher relative risk at low doses as opposed to high doses. Pierce, et al., at p. 9.

ORDER RE SUMMARY JUDGMENT-   403

1   Plaintiffs do not dispute what defendants represent is the

2   thinking of ICRP, NCRP and BEIR V regarding the use of a DREF.

3   With regard to "Risk Assessment," BEIR V states:

4   Since the risk models were derived primarily
          from data on acute exposures . . . the application

5   of these models to continuous low dose-rate
          exposures requires consideration of the dose rate

6   effectiveness factor (DREF) . . . .

7   (BEIR V at p. 171). However, plaintiffs note that BEIR V goes on

8   to say:

9   For the leukemia data [non-solid cancer], a linear
          extrapolation indicates that the lifetime risks

10   per unit bone marrow dose may be half as large for
          continuous low dose rate as for instantaneous high

11   dose rate exposures. **For most other cancers . . . the
          estimated DREFs are near unity.** Nevertheless, the

12   committee judged that some account should be taken
          of dose rate effects and . . . suggests a range of

13   dose rate reduction factors that may be applicable.

14   (Id. at pp. 171 and 174)(Emphasis added).

15   Based on the foregoing, the court concludes that, as in the

16   iodine case, this matter of application of a DREF is subject to

17   legitimate scientific debate.[310] Accordingly, the doubling

18   doses set forth above (and below) are not reduced to account for

19   a DREF.

20

21   **(3)   Remaining Cancer Claims**

22   The remaining cancers include stomach, colon, rectal,

23   pancreatic, non-melanoma skin, breast (lactating female and non-

24   _____

25   [310] Leukemia is not considered a "solid" cancer. Therefore,
     in the absence of data showing the DREFs are also "near unity"

26   for leukemia, application of a DREF may be more compelling for
     that condition. However, the court still believes there is an

27   issue of material fact even with regard to leukemia and DREF.

28   **ORDER RE SUMMARY JUDGMENT-   404**

1 lactating female), ovarian, testicular, urinary tract and kidney,

2 nervous system (brain), liver, bone, lung (including trachea and

3 bronchus), and leukemia (excluding chronic lymphocytic leukemia).

4   It does not appear defendants specifically challenge whether

5 radiation is "capable of causing" these particular cancers.[311]

6 Defendants are willing to accept the risk co-efficients contained

7 in Radford's non-iodine **report** for the purpose of computing the

8 doubling doses for each of these cancer sites.   The risk co-

9 efficients are found at pp. 17-18 of Radford's Non-Iodine Report.

10 The doubling doses as computed by defendants from the risk co-

11 _____

       [311] They do assert that for rectal, pancreatic and kidney
12 cancer, the results from the Thompson study are not statistically
   significant and there was no finding of radiation-related excess
13 of cancer. Defendants' Opening Br. at p. 29, citing Thompson, et
   al., 1994 at S17. As noted above, plaintiffs contend
14 "statistical significance" is no longer of consequence in the
   radiation/cancer causation context.
15   Defendants point out that Thompson did not find a radiation
   effect for cancers of the brain. It did not report a
16 statistically significant result. (Thompson, et al., 1994 at
   S57). However, Radford relied on data from BEIR V in deriving
17 his 3.25 risk co-efficient. (Radford Dep. at pp. 159-63).
       Defendants claim that for non-melanoma skin cancer, Radford
18 testified the epidemiological data show there is a threshold dose
   of 100,000 millirem for induction of such cancer. (Radford Dep.
19 at p. 153). In other words, below that dose there is no evidence
   radiation is "capable of causing" non-melanoma skin cancer.
20 Plaintiffs contend there is at least an issue of material fact on
   the existence of a threshold due to evidence showing, in general,
21 a **no-threshold** dose response for radiation and cancer. They also
   note that Radford testified the Thompson study found the non-
22 melanoma data "fit just as well" on the linear (no-threshold)
   dose response curve as on the alternative dose response model
23 involving a "linear spleen." Radford testified the "linear
   spleen" model was consistent with a threshold of 1 Sievert (100
24 rem; 100,000 millirem). (Radford Dep. at p. 153).
       There appears to be at least an issue of material fact
25 whether there is a threshold for non-melanoma skin cancer, but
   the dispute is irrelevant since the doubling dose is also 100,000
26 millirem. Anyone exposed to 100,000 millirem or less will be
   dismissed because they cannot show radiation was a "more likely
27 than not" cause of their non-melanoma skin cancer.

28 **ORDER RE SUMMARY JUDGMENT-   405**

1 efficients are found at p. 32 of Defendants' Opening Brief re

2 Non-Iodine. A summary of the risk co-efficients and the doubling

3 doses is as follows:

4

5 | Cancer Site or Type | ERR/Sv (100 rem)[312] | Doubling Dose[313] |
   |---|---|---|
6 | Stomach | 0.32 | 313,000 |
7 | Colon | 0.72 | 139,000 |
8 | Rectum | 0.21 | 476,000 |
9 | Pancreas | 0.18 | 556,000 |
10 | Skin (Non-Melanoma) | 1.0 | 100,000 |
11 | Breast (Female) | 1.59 | 63,000 |
12 | Ovary | 0.99 | 101,000 |
13 | Urinary Tract and Kidneys | 1.24 | 81,000 |
14 | Nervous System (Brain) | 3.25 | 31,000 |
15 | Testes | 3.44 | 29,000 |
16 | Liver | 0.49 | 204,000 |
17 | Bone | 0.40 | 250,000 |
18 | Lung | 0.95 | 105,000 |
19 | Leukemia | 4.50 | 22,000 |

20

21   The plaintiffs argue it is inappropriate at this time to use

22 Radford's "average" risk co-efficients for the purpose of

23 _____

24   [312] Without increase for individual susceptibility factor.
   Radford provides a second set of risk co-efficients incorporating
   his individual susceptibility factor which has the effect of
25 increasing the co-efficients by a factor of five, while
   decreasing the doubling doses by a factor of five.

26
     [313] Expressed in millirems. Does not include a decrease for
27 Radford's individual susceptibility factor.

28
   ORDER RE SUMMARY JUDGMENT-   406

1 computing generic doubling doses. According to plaintiffs, **"in**

2 **general**, [Radford's] risk co-efficients do not yet take into

3 account individual factors affecting risk, including, among

4 others, age at exposure and gender."[314] Plaintiffs cite

5 Radford's non-iodine report in which he states:

6   In the preceding text, I have identified trends
         of risk with age, and differences by sex. In
7   general, women are at greater risk than men,
         although in a few instances the effect is
8   opposite. Also young children are generally at
         much greater risk than older persons. A detailed
9   application of the A-bomb results therefore
         requires both age and sex to depict the cancer
10   risk adequately. For some of the cancers, the
         statistical variation of risk by age is sufficiently
11   large that smoothing of the relationship of age to
         ERR/Sv is appropriate. The age and sex effects have
12   not been included in the following table [table of
         risk co-efficients], but they will be when considering
13   individual cases.

14 (Radford 1996 Non-Iodine Rpt. at p. 16).

15   Radford stated that although his "average values" were for

16 "all ages and sexes," it would be necessary in "individual cases"

17 to consider age and sex "in modifying the . . . risk co-

18 efficients, for the purposes of determining causation." (_Id_. at

19 p. 17).

20   According to plaintiffs, Radford will determine the

21 appropriate risk co-efficients at the individual causation stage

22 of the proceedings, including, for example, a risk co-efficient

23 _____

24   [314] Elsewhere, plaintiffs refer to the risk co-efficients
  listed in Radford's report as "merely benchmarks, combining risks
25 for all persons at all ages into a single risk number for a
  particular cancer site." Plaintiffs add that the risk co-
26 efficients are "'points of departure' ill-suited for determining
  causation without further individualized analysis." (Plaintiffs'
27 Joint Response at pp. 73-74).

28 **ORDER RE SUMMARY JUDGMENT-   407**

1 for the 0-9 age group insofar as liver cancer. At his

2 deposition, Radford testified as follows:

      Q: So for that person [exposed at age 9 and under],
3   you are not able to render an opinion that
           [his/her] liver cancer was caused by radiation?
4

      A: We have no evidence from the A-Bomb studies,
5   but what I would do would be to look at some of
           the other study populations in which liver cancers
6   have been found in excess, such as, for example
           the Thorotrast patients. You look at all the data
7   as best you can and observe it and base an opinion
           on it.
8

9 (Radford Dep. at p. 106).

10   All of this hearkens back to an issue which the court has

11 already addressed to some extent: is it appropriate to use

12 generic doubling doses (based on a population baseline risk), or

13 must one wait to develop individual doubling doses (based on an

14 individual baseline risk)?

15   The defendants argue Radford provides all of the details

16 necessary to determine the doubling doses for each of the cancers

17 at issue, and that by failing to propose alternative risk co-

18 efficients, the plaintiffs have forfeited their right to contest

19 the "values" presented in defendants' opening brief (i.e. the

20 risk co-efficients and doubling doses set forth above).

21 The plaintiffs, of course, argue that risk co-efficients are

22 wholly irrelevant to what they define as their burden of proof at

23 this stage of proceedings (is radiation "capable of causing"

24 cancer?). Plaintiffs say they were under no obligation to

25 provide risk co-efficients at this stage of the proceedings.

26 (See n. 47 at p. 73 of Plaintiffs' Joint Response Brief).

27   The court disagrees with plaintiffs' assertion as to the

28 **ORDER RE SUMMARY JUDGMENT-   408**

1   applicable evidentiary standard. Therefore, the question becomes

2   whether even under a "doubling of risk" standard ("more likely

3   than not"), it is appropriate to use generic doubling doses,

4   rather than waiting to calculate individual doubling doses. The

5   court is persuaded it is appropriate to employ generic minimum

6   doubling doses for the purpose of determining which claims should

7   proceed to the individual stage, and perhaps trial.

8   At his deposition, Radford acknowledged his risk co-

9   efficients might require adjustment to account for gender and age

10   at the time of exposure. He also acknowledged his proposal to

11   make an adjustment for individual susceptibility for persons

12   "more susceptible" to cancer- a five factor upward adjustment of

13   the risk co-efficients. (Radford Dep. at pp. 91-92). He was

14   then asked:

15   Q: **Are there any other adjustments that you propose
                 to make for the risk co-efficients?**

16

17   A: **Those were the principal ones, yes.**

18   (Id. at p. 92)(Emphasis added). Neither in his report, or in his

19   deposition, did Radford identify any other specific adjustments,

20   with the exception of smoking and lung cancer.

21   Defendants point out that for some of the cancers, Radford

22   specifically indicated the risk co-efficient could be used for

23   all ages and both genders- i.e. gallbladder (Radford Dep. at p.

24   146) and bone cancer (Radford Dep. at p. 172- discussed infra).

25   And in other cases, Radford made specific adjustments to take

26   into account age and gender differences- i.e. esophageal cancer

27   (0.25 ERR/Sv for males and 0.35 ERR/Sv for females **over the age**

28

ORDER RE SUMMARY JUDGMENT-   409

1 **of 20**); non-Hodgkin's lymphoma (.60 ERR/Sv for males; no ERR/Sv

2 for females).

3   The Thompson A-Bomb study shows Radford had all the age and

4 gender data he needed. If he wanted to carve out particular age

5 and gender categories, he could easily have done so. By using an

6 "average" ERR/Sv figure based on those categories, Radford may

7 have decreased the doubling dose applicable to a particular age

8 and gender category, but at the same time, he **increased** the

9 doubling dose otherwise applicable to another particular age and

10 gender category.

11   For example, based on the Thomspon A-bomb data, the average

12 ERR/Sv for colon cancer is 0.72 with a corresponding doubling

13 dose of 139,000 millirem. For males ages 0-9 at the time of

14 exposure, the ERR/Sv is 2.39 which produces a doubling dose of

15 42,000 millirem. However, for males age 40 and over at the time

16 of exposure, the ERR/Sv is 0.30 with a corresponding doubling

17 dose of 333,000 millirem. (Table XXVI, Defendants' Ex. 218 at p.

18 5). Use of an "average" figure might save the claim of a 41 year

19 old man with colon cancer, whereas that would not be the case if

20 the ERR/Sv for his particular age and gender category was used.

21   Radford's proposed individual susceptibility factor supports

22 the notion that generic minimum doubling doses (derived from

23 population based epidemiological studies) are appropriate.

24 Radford is willing to apply this factor to **all** of his risk co-

25 efficients. He is willing to apply it to **all** individuals,

26

27
28 **ORDER RE SUMMARY JUDGMENT-   410**

1 regardless of age, gender and level of susceptibility[315], for

2 the alleged purpose of providing a "point of departure" from

3 which to make even further potential upward adjustments at the

4 individual stage. However, the court must question whether any

5 further upward adjustments would be justified since doing so

6 would merely duplicate what was already considered in applying

7 the increased susceptibility factor.

8   Assuming plaintiffs were content to live with the doubling

9 doses generated by use of Radford's individual susceptibility

10 factor, the result would be **"generic minimum doubling doses."**

11 Thus, at the same time the plaintiffs argue it is inappropriate

12 to set generic doubling doses, it appears they are proposing to

13 establish their own such doses provided an individual

14 susceptibility factor is included.

15   The fact Radford will not be allowed to employ his

16 individual susceptibility factor does not mean, however, that

17 generic minimum doubling doses are inappropriate. The

18 epidemiological studies from which Radford derives his risk-

19 coefficients (in particular, the A-Bomb study) include subjects

20 representing the entire spectrum of the exposed population and

21 therefore, the entire spectrum of susceptibilities (due to diet,

22 smoking, genetic make-up, etc.). In other words, susceptibility

23 is already taken into account and therefore, an "individual

24 _____

25   [315] This is one of the major reasons for striking Radford's
   individual susceptibility factor. The literature from which he
26 purportedly derives his factor points out that some individuals
   are actually **less** susceptible. Yet Radford proposes only an
27 **upward** adjustment.

28 **ORDER RE SUMMARY JUDGMENT-   411**

1   susceptibility factor" is unnecessary.

2   According to Radford:

3   Where comparisons of results of radiation
               exposure have been made between the A-bomb
4   survivors and Western populations **the results
               appear to be reasonably consistent with
5   each other.** A high proportion of Japanese
               men are cigarette smokers, but relatively
6   few Japanese women smoke. Obviously also
               the diet in Japan differs in many ways from
7   common diets in other countries. For all
               these reasons, care must be taken in applying
8   the Japanese results [to] other populations,
               but for some cancers the Japanese data are
9   the only source available. In this report,
               the A-bomb data have been emphasized because
10   they are the most comprehensive that we have.

11   (Radford Rpt. at p. 7)(Emphasis added).

12   Although Radford suggests a potential distinction in diet,

13   he did not alter his risk co-efficients on that basis. He did

14   not re-analyze the epidemiological data for the purpose of

15   showing a higher risk co-efficient was warranted for a particular

16   cancer (i.e. colon) due to a Western diet.[316] Nor, say

17   defendants, could he because the epidemiological studies from

18   which he derives his risk co-efficients (the A-bomb data) do not

19   analyze the role of diet on radiation risk, or provide risk co-

20   efficients that vary according to diet.

21   At his deposition, Radford discussed the impact of smoking

22   in connection with lung cancer. Based on certain epidemiological

23

24   _____

25   [316] In Daubert II, the Ninth Circuit observed that because
     the plaintiffs' experts did not seek to differentiate the
26   plaintiffs from the subjects of the statistical studies, "[t]he
     studies must therefore stand or fall on their own." 43 F.3d at
27   1321, n. 16.

28

ORDER RE SUMMARY JUDGMENT-   **412**

data[317], Radford testified the relative risk for non-smokers from radiation exposure is about three times higher than it is for smokers. (Radford Dep. at p. 151). Radford provided specific risk co-efficients reflecting the difference. Smoking was identified as a factor by Radford only in connection with lung cancer. Therefore, where he felt it was appropriate, Radford made adjustments to his risk co-efficients based on smoking, age and gender.

Plaintiffs have available to them right now, all of the epidemiological data necessary to derive risk co-efficients taking into account age, gender, smoking and any other factor. The individual risk analysis proposed by the plaintiffs is dependent on the same **population based** epidemiological studies which already exist.[318] Whether the individual factors cited by plaintiffs- diet, smoking, genetic susceptibility, medical history- increase or decrease the risk that radiation exposure is a "more likely than not" cause of cancer must be established by epidemiological evidence.

Plaintiffs repeatedly refer to medical experts taking the stand and providing differential diagnoses which they claim will isolate radiation as a "more likely than not" cause of cancer in

---

[317] An epidemiological study which Radford is conducting with regard to the incidence of lung cancer in Swedish iron miners exposed to radon. (Radford Dep. at pp. 19-20).

[318] Radford says that with regard to liver cancer and individuals exposed at ages 0-9, he will look at data other than the A-bomb study- the Thorotrast patients- for the purpose of evaluating risk. This is something Radford should have already examined if it somehow impacts the risk co-efficients for liver cancer.

ORDER RE SUMMARY JUDGMENT-   413

a particular individual. Thus, a doctor might testify an

individual plaintiff has a low fat diet, does not smoke, has

no genetic susceptibilities, and has nothing else in his/her

medical which might account for his/her cancer. Nonetheless,

because radiation-induced cancers cannot be distinguished from

any other cancers, the doctor's testimony by itself is not

sufficient to sustain a jury verdict in the absence of

epidemiological data raising an inference radiation is a "more

likely than not" cause of the individual's cancer.[319]

(a) Liver Cancer

At his **deposition**, Radford testified he could not assign an

excess relative risk for liver cancer to children exposed between

ages 0 and 9. According to Radford, there was no evidence of

excess liver cancer in that group over a period of 45 years.

(Radford Dep. at p. 109).[320] In the absence of a risk co-

efficient, defendants suggest the court can dismiss all liver

---

[319] The epidemiological data will allow an inference that
radiation is a "more likely than not" cause in individuals of a
particular age and gender. Where the data is **available**, it may
also allow for such an inference in individuals with a particular
type of diet, smoking history, genetic susceptibility, and
medical background. The fact Radford appears not to have cited
any epidemiological data taking into account diet, genetic
susceptibility and medical background may be due to the fact none
exists in the radiation context. Of course, even if such data
existed, there would have to be some similarity between the study
population and the individuals or population for whom
extrapolation of the study results is sought.

[320] Indeed, the table from Thompson 1994, at S39 (Table
XXXII) shows why: the ERR/Sv for both males and females in the 0
to 9 group is **-0.25.** (Table is reprinted at p. 11 of Defendants'
Ex. 218).

ORDER RE SUMMARY JUDGMENT-   414

1 cancer claims based on exposures occurring between ages 0 and 9.

2 Simply put, this is because there is no evidence from which a

3 doubling of risk can be inferred.

4   Plaintiffs contend the defendants have drawn an "incorrect

5 and inappropriate" inference from Radford's deposition testimony

6 that there is no evidence radiation is "capable of causing" liver

7 cancer in persons exposed between ages 0-9. However, the concern

8 is not whether radiation is "capable of causing" liver cancer in

9 persons exposed between ages 0-9. It is whether there is any

10 evidence from which it can reasonably be inferred that radiation

11 is a "more likely than not" cause of liver cancer in such

12 persons.

13   Plaintiffs admit they do not have any such evidence at this

14 time but contend, as discussed above, they will develop such at

15 the individual causation stage. For reasons set forth above,

16 there is no compelling reason to wait. If plaintiffs do not have

17 the necessary epidemiological evidence now, there is no reason to

18 expect they will have it later on. One can only speculate

19 whether additional, material epidemiological studies will be

20 developed. Accordingly, based on Radford's testimony, the court

21 will dismiss all liver cancer claims by those individuals ages 0-

22 9 at the time of exposure. There is an absence of expert proof

23 from which an inference can be drawn that radiation is a "more

24 likely than not" cause of liver cancer in such individuals.

25   At his deposition, Radford acknowledged that for women

26 exposed between ages 10 and 19, Thompson reported an ERR/Sv for

27 liver cancer of -0.25 as compared to 1.38 for males. (See

28

ORDER RE SUMMARY JUDGMENT-   415

Defendants' Ex. 218 at p. 11). Radford testified that for females in the 10-19 group, he would "assign a small risk co-efficient, using the average of 0.17 [ERR/Sv]" for females across all ages. (Radford Dep. at pp. 106-07). An ERR/Sv of 0.17 results in a doubling dose of 588,000 millirem.[321]

(b)   Bone Cancer

In his non-iodine report, Radford proposed a risk co-efficient of 0.40 ERR/Sv for bone cancer. (Radford Rpt. at p. 18). At his deposition, he changed the figure to 0.60. Radford testified the 0.40 figure was based on evidence available to him at the time he prepared his report, specifically Darby, et al., "Long-term mortality after a single treatment course with X-rays in patients treated for ankylosing spondylitis," **Br. J. Cancer** 55: 179-190 (1987).[322] According to Radford, the newer Pierce study reports an excess of bone cancer among A-Bomb survivors: .88 ERR/Sv for males and .75 ERR/Sv for females. Based on that, Radford arrived at an average figure of .80. He then averaged the .80 figure with the .40 figure from Darby to arrive at 0.60 which he is willing to apply "across the board" to all ages and genders. (Radford Dep. at pp. 171-72).

Defendants argue Radford "improperly" changed his report.

---

[321] Defendants apparently are willing to accept this figure (588,000 millirem) even though no excess is reported for this age and gender category (females ages 10-19 at the time of exposure). The risk co-efficient (0.17 ERR/Sv) from which this doubling dose is derived is less than the average co-efficient (0.49) found in Radford's report.

[322] Plaintiffs' Ex. 33 to Appendix 4 re Non-Iodine Claims.

ORDER RE SUMMARY JUDGMENT-   416

1   They do not, however, challenge the change on substantive

2   scientific grounds. The requirement of an expert report is

3   intended to prevent experts from becoming "moving targets" with

4   regard to their opinions. At the time Radford issued his non-

5   iodine report (March 1996), Pierce was not available. It was

6   available at the time of his deposition which commenced in

7   November 1996. Defendants had an opportunity to depose Radford

8   on his revised risk co-efficient. Radford discussed his revised

9   figure in the November 1996 segment of his deposition. His

10 deposition resumed and concluded in February 1997. Therefore,

11 defendants and their experts had an opportunity to review the

12 Pierce study in the interim and ask Radford any additional

13 questions about his new bone cancer figure. Based on these

14 facts, the court fails to see the type of prejudice which would

15 warrant striking the new figure because it was not included in

16 Radford's expert report.[323]

17   In any event, defendants contend 0.60 ERR/Sv generates a

18 doubling dose (167,000 millirem) which exceeds the dose received

19 by any of the plaintiffs.

20

21   **(c) Lung Cancer (Including Trachea and Bronchus)**

22   In his report, Radford proposed a risk co-efficient of 0.95

23 ERR/Sv. As noted above, at his deposition, Radford testified he

24 would use different risk co-efficients for smokers versus non-

25 smokers. He proposes a risk co-efficient of 1.3 ERR/Sv for non-

26 _____

    [323] If Radford had waited until his declaration to change

27 the figure, that would be a different issue.

28 **ORDER RE SUMMARY JUDGMENT-   417**

1 smokers, which corresponds to a doubling dose of 77,000 millirem.

2 For smokers, he proposes a risk co-efficient of 0.40, which

3 corresponds to a doubling dose of 250,000 millirem. (Radford

4 Dep. at pp. 151-52). The defendants do not challenge these

5 figures.

6   As with liver cancer, Radford acknowledges the Thompson A-

7 Bomb study did not report any excess for lung cancer in the group

8 ages 0-9 at the time of exposure.[324] (Id. at 152). Once again,

9 plaintiffs argue that at the individual stage, Radford will come

10 up with a risk co-efficient for individuals in this age group.

11 For reasons set forth above, there is no compelling reason to

12 wait. If there is not enough data to generate a risk co-

13 efficient now, the court fails to see how there will be enough

14 data at a later time. It is pure speculation that some study may

15 materialize in the interim. Furthermore, the court must put an

16 end to development of the expert record or this case will go on

17 forever.

18   Plaintiffs also cite the ill-fated R-11 Study as showing the

19 risk of lung cancer is increased three-fold among persons who

20 went to high school downwind of the Hanford facility. The R-11

21 Study is not scientifically reliable. Besides that, it is not

22 relevant to the assessment of risk because it provides no

23 information about dose. The raw incidence of disease says

24 nothing about risk unless it is tied to radiation dose.

25 _____

26   [324] See Table XLVI of Thompson reprinted at p. 27 of
Defendants' Ex. 218. All of the figures for the 0-9 group are in

27 the negative (-0.26 for both males and females).

28 **ORDER RE SUMMARY JUDGMENT-   418**

The court will dismiss all trachea, bronchus, and lung

cancer claims by those individuals ages 0-9 at the time of

exposure. There is an absence of expert proof from which an

inference can be drawn that radiation is a "more likely than not"

cause of trachea, bronchus, and lung cancer in such individuals.

        (d) Leukemia

    In his non-iodine report, Radford provided an "approximate"

ERR/Sv of 4.5. This figure is derived from a table in the

Preston study providing relative risk figures for males and

females in three different age categories: 0-19; 20-39; and 40

or more. (Radford Rpt. at p. 15). Radford indicated this data

might have to be modified somewhat, depending on age. He stated

those exposed below age 20 have twice the risk as those exposed

at older ages, while "[e]arlier A-bomb analyses have shown that

those exposed at ages less than ten are even at a higher risk."

(Id.)

    At his deposition, Radford reiterated that age at the time

of exposure is very important in assessing risk for leukemia.

(Radford Dep. at p. 173). He indicated that for somebody who was

1 year old when they were irradiated and contracted leukemia at

about age 10, the risk could be "3 times higher." Radford was

asked whether this would equate to an ERR/Sv of about 13.5 (4.5 x

3), to which his response was "these are approximate numbers."

(Id. at p. 177). According to defendants, a 13.5 ERR/Sv results

in a doubling dose of 7,000 millirem.

    The court will not allow Radford to use the 13.5 figure.

ORDER RE SUMMARY JUDGMENT-   419

1   Radford opted for an "average" figure which spans all age

2   categories and pertains to both sexes. While that may have the

3   effect of increasing the doubling dose for children, it has the

4   effect of decreasing the doubling dose for older individuals,

5   particularly males, for whom the relative risk figures are

6   significantly lower.

7

8   **c.   Conclusion**

9   Based on the foregoing:

10   1)   The court will grant summary judgment and dismiss with

11   prejudice all non-cancer claims resulting from alleged exposure

12   to non-iodine emissions.

13

14   2)   The court will grant summary judgment and dismiss with

15   prejudice all plaintiffs' claims for Hodgkin's disease, cervical

16   cancer, uterine cancer, melanoma skin cancer, and chronic

17   lymphocytic leukemia.

18

19   3)   The court will grant summary judgment and dismiss all

20   claims: a) by female plaintiffs for Non-Hodgkin's lymphoma; and

21   b) by female plaintiffs for Esophageal Cancer who are 20 years

22   old or younger and **currently suffering** from that condition.

23

24   4)   The court will grant summary judgment and dismiss with

25   prejudice all plaintiffs' claims for:

26   a.   Prostate cancer based on exposure to 345,000 millirem or

27   less;

28

ORDER RE SUMMARY JUDGMENT-   420

b.   Non-Hodgkin's Lymphoma (**Males Only**) based on exposure to 167,000 millirem or less;

c.   Gallbladder Cancer based on exposure to 833,000 millirem or less;

d.   Nasal Cavity Cancer based on exposure to 455,000 millirem or less;

e.   For males, Esophageal Cancer based on exposure to 400,000 millirem or less; For females ages 21 and over, Esophageal Cancer based on exposure to 286,000 millirem or less;

f.   Cancer of the Oral Cavity and Pharynx based on exposure to 345,000 millirem or less;

g.   For adults (presumably ages 20 and over), Salivary Gland Cancer based on exposure to 33,000 millirem or less; For children (presumably ages 10-19), Salivary Gland Cancer based on exposure to 17,000 millirem or less; For infants (presumably ages 0-9), Salivary Gland Cancer based on exposure to 10,000 millirem or less.[325]

5)   Considering the modifications made by Radford at his deposition, the court will grant summary judgment and dismiss with prejudice all plaintiffs' claims for:

a.   Stomach Cancer based on exposure to 313,000 millirem or less;

b.   Colon Cancer based on exposure to 139,000 millirem or less;

_____

[325]   The age breakdown is based on the ages used in the Thompson study.

ORDER RE SUMMARY JUDGMENT-   421

  c. Rectal Cancer based on exposure to 476,000 millirem or less;

  d. Pancreatic Cancer based on exposure to 556,000 millirem or less;

  e. Non-Melanoma Skin Cancer based on exposure to 100,000 millirem or less;

  f. Breast Cancer (Female) based on exposure to 63,000 millirem or less;[326]

  g. Ovarian Cancer based on exposure to 101,000 millirem or less;

  h. Cancer of the Urinary Tract and Kidneys based on exposure to 81,000 millirem or less;

  i. Cancer of the Nervous System (Brain) based on exposure to 31,000 millirem or less;

  j. Testicular Cancer based on exposure to 29,000 millirem or less;

  k. Liver cancer based on exposure at **age 10 or over** to 204,000 millirem or less, with the exception of females ages 10-19 for which exposure must exceed 588,000 millirem. **All liver cancer claims by individuals ages 0-9 at the time of exposure will be dismissed.**

  l. Bone cancer based on exposure to 167,000 millirem or

---

[326] For lactating females only, the exposure can be a mixture of iodine and non-iodine exposure. For non-lactating females, the exposure is based solely on non-iodine emissions (plutonium).
Other cancers for which a combination of iodine and plutonium is relevant include salivary gland cancer, stomach cancer, and bladder cancer.

ORDER RE SUMMARY JUDGMENT- 422

1 less;

2   m.   For **non-smokers**, Trachea, Bronchus and Lung cancer based

3 on exposure at **age 10 or over** to 77,000 millirem or less; for

4 **smokers**, exposure at **age 10 or over** to 250,000 millirem or less.

5 **All trachea, bronchus and lung cancer claims by individuals ages**

6 **0-9 at the time of exposure will be dismissed.**

7   n.   Leukemia (excluding chronic lymphocytic variety) based

8 on exposure to 22,000 millirem or less.

9

10   Only those cancer claims based on exposures above these

11 doubling doses can conceivably proceed into individual causation

12 discovery and perhaps to trial. Defendants assert, however, that

13 no plaintiff can show he/she was exposed to an organ dose

14 exceeding any of these doubling doses and consequently, that no

15 claims can proceed to Phase III. That is the next issue to be

16 addressed.

17

18   **2.   Dose**

19   **a.   Klementiev Plutonium Source Term Analysis**

20   **(1) Introduction**

21   Dr. Klementiev has submitted three non-iodine (plutonium)

22 reports in this case. The first is dated March 30, 1996,

23 entitled "Estimation Of Pu-239 Releases To The Atmosphere From

24 The Hanford Site." A supplemental report, "Estimation Of Pu-239

25 Releases To The Atmosphere From The Hanford Site: New Findings,"

26 is dated October 4, 1996. Defendants filed a motion to strike

27 this supplemental report on the basis that it did not meet the

28 **ORDER RE SUMMARY JUDGMENT-   423**

1 court's supplementation criteria.

2   In a December 16, 1996 order (Ct. Rec. 878), the court

3 denied the motion to strike. The court also allowed the

4 plaintiffs additional time to sift through alleged newly

5 discovered documents pertaining to plutonium releases from

6 Hanford, and to submit additional supplemental reports based on

7 those documents no later than March 3, 1997. The court made it

8 clear that any such reports still needed to comply with the

9 court's supplementation criteria. Klementiev's February 28, 1997

10 report, "Estimation Of Pu-239 Releases To The Atmosphere From The

11 Hanford Site: Uncertainty Modeling," was submitted as a

12 supplemental report pursuant to the court's December 16, 1996

13 order.

14   Defendants move to exclude all of Klementiev's plutonium

15 reports on the basis that he is not qualified to render the

16 opinions he has offered, those opinions are not scientifically

17 reliable (i.e. are the result of unsound scientific methodology),

18 and do not "fit" the relevant causation inquiry. Because

19 plaintiffs' experts Douglas Stewart and Douglas Crawford-Brown

20 rely on Klementiev's release estimates, defendants contend

21 exclusion of their reports is also necessitated. Stewart uses

22 Klementiev's release estimates to calculate concentrations of

23 plutonium in the air. Crawford-Brown uses those air

24 concentrations to calculate plutonium doses.

25

26   **(2) Overview of Plutonium Production Process**

27   The parties agree on the basics of how the plutonium

28 **ORDER RE SUMMARY JUDGMENT-   424**

1 production process took place at Hanford. First, uranium metal

2 rods were placed inside a reactor. Nuclear reactions occurred

3 which converted some of the uranium into plutonium. Second, the

4 plutonium was separated from the fuel rods by chemical means.

5 The fuel rods were brought to a **separations plant** and dissolved

6 in nitric acid. The resulting solution was chemically processed

7 until all that remained was relatively pure plutonium nitrate.

8 The third step was conversion of the plutonium nitrate into

9 plutonium metal.[327] This took place in the **Plutonium Finishing**

10 **Plant (PFP)** (also known as the 234-5Z Plant) which, like the

11 **separations plants**, was located in the "200 Area" of Hanford.

12   The finishing process involved various phases where the

13 plutonium nitrate was melted and cast into various shapes for use

14 in weapons. One phase was "Reduction" or "Burning" where

15 plutonium tetrafluoride was mixed with calcium, gallium, and

16 other agents, then fired at very high temperatures until fused

17 into plutonium metal "buttons." Another phase was "Casting"

18 where the "buttons" were melted and poured into semi-spherical

19 molds. These processes were conducted in vacuum glove boxes

20 connected to a plant-wide ventilation and filtration system.

21

22   **(3)   HEDR Analysis of Plutonium Emissions**

23   It is appropriate to begin with HEDR's analysis of plutonium

24 emissions from Hanford since Klementiev's plutonium analysis

25 essentially is a response to, and a critique of, the HEDR

26 ───────────────

27   [327] This step did not commence until 1949.

28 **ORDER RE SUMMARY JUDGMENT-   425**

1 analysis.

2   HEDR first estimated the amount of plutonium processed at

3 the Hanford separations plants.[328] To determine how much

4 plutonium was released through the four separations plant vent

5 stacks, HEDR used stack measurement data to derive "release

6 fractions." C.M. Heeb, "Radionuclide Releases to the Atmosphere

7 from Hanford Operations, 1944-1972" (1994), p. 4.27 (hereinafter,

8 "Heeb (1994)").[329] Applying these fractions to the amount of

9 plutonium processed, HEDR determined that plutonium releases from

10 the separations plants totaled 1.78 curies (Ci), or about 29

11 grams, between 1944 and 1972.[330] (Id. at p. vii of "Preface").

12   In another report, Heeb and Gydesen, "Sources of Secondary

13 Radionuclide Releases from Hanford Operations," (1994) (PNWD-2254

14 HEDR)[331], HEDR examined emissions from the Z-Plant. The PFP or

15 234-5Z Plant was equipped with a 200 foot vent stack known as the

16 291-Z stack.[332] HEDR found the releases from the Z-Plant were

17 smaller than from the separations plants. It did not detail what

18 it considered to be the specific amount of plutonium released.

19 ───────────────────

20   [328] There were four different separations plants known as
    the T Plant, the B Plant, REDOX, and PUREX.

21   [329] Defendants' Ex. 49.

22   [330] A curie is a measure of radioactivity, whereas a gram is
    a measure of physical weight. One curie of Pu-239 is equal to
23 about 16 grams (0.016 kilograms).

24   [331] Defendants' Ex. 170.

25   [332] The Z-Plant is actually a collective term for five
    different buildings which were involved in plutonium processing,
26 including 234-5Z (PFP), 231-Z, 232-Z, 236-Z and 242-Z. The 231-Z
    plant (plutonium machining and metallography) had a separate
27 exhaust stack, the 231-Z stack.

28 **ORDER RE SUMMARY JUDGMENT-   426**

1   HEDR estimated that the highest cumulative plutonium dose

2   resulting from the release of the 1.78 curies from the

3   separations plants was **2.5 millirems EDE** (Effective Dose

4   Equivalent). This dose estimate assumes the exposed person lived

5   continuously at Ringold- the maximum dose location in the HEDR

6   study area- for the entire period from 1945 to 1972. Farris, et

7   al., "Atmospheric Pathway Dosimetry Report, 1944-1992," (1994),

8   at Appendix C, Table C.7.[333]

9

10   **(4) Klementiev's Analysis**

11   In his March 1996 report, Klementiev analyzed the following

12   "Pu-239 emitters:" 1) separations plant stacks; 2) 100 area

13   reactor stacks; 3) 224 concentration building roof fans; and 4)

14   Z-plant stack. His October 1996 report was restricted to the Z-

15   Plant. Based on "new information" he received after his October

16   1996 report, Klementiev undertook in his February 1997 report:

17   1) a reassessment of the estimates of airborne Pu-239 presented

18   in his previous reports (March and October 1996); and 2) an

19   uncertainty analysis of the airborne Pu-239 releases presented in

20   his previous reports. According to Klementiev, the uncertainty

21   modeling altered his mean values of the estimated releases.

22   (Klementiev 1997 Rpt. at p. 2).

23   Defendants' motion in limine focuses on Klementiev's release

24

25

26   ───────────────────

27   [333]   Defendants' Ex. 29.

28
     ORDER RE SUMMARY JUDGMENT-   427

1   estimates for the **Z-Plant**.[334] Klementiev's first estimate is

2   0.15 (2.4 g) Ci which is based on what he refers to as the

3   "source spreadsheet data." This data is actually the historical

4   measurements of plutonium emissions taken from the Z-Plant

5   exhaust stack (291-Z), also known as "stack-sampling data."

6   (Klementiev Dep. at p. 448).[335] 0.15 Ci represents a mean value

7   in a 95% certainty range of 0.1 Ci to 0.22 Ci. (Klementiev 1997

8   Rpt. at p. 6 and App-13).

9   Klementiev's second estimate is based on historical PFP

10   plutonium inventory records, specifically Anderson, J.D., "A

11   Study of 234-5 Building Inventory Difference for the Years 1956

12   through 1966," (1977) (hereinafter "Anderson 1977"). Anderson

13   reported that a "600 kilogram inventory difference associated

14   _____

[334] Defendants do not concern themselves with Klementiev's
estimate for plutonium releases from the separations plants and
any other areas, contending the amounts are too small to generate
doses anywhere near approaching the necessary doubling doses.
    In his March 1996 report, the highest estimate produced by
Klementiev for the separations plants, based on his various
scenarios, was 40 Ci (curies) between 1944 and 1972. (Klementiev
March 1996 Rpt. at pp. 12-13, 16). Klementiev considered the
separations plants in his February 1997 uncertainty analysis.
According to defendants, Klementiev's final release estimate is
44.44 curies.
    For the concentration facilities (224 concentration building
roof fans), Klementiev reported a mean estimate of 36.7 Ci based
on his uncertainty analysis. (Klementiev 1997 Rpt. at p. 8 and
App. 14).
    For the 100 Area reactor stacks, Klementiev concluded in his
March 1996 report that the releases were "negligible" and so he
did not include them in any further analysis. (Klementiev March
1996 Rpt. at p. 13).
    As will be apparent, Klementiev's estimates for the
separations plants and the concentration facilities do not
produce doses exceeding the applicable doubling doses.

[335] This is the same type of data HEDR used to estimate
releases from the **separations plants**.

ORDER RE SUMMARY JUDGMENT-   428

1 with operations in the 234-5 Building, developed during the

2 period 1956 to 1966." Attached to the Anderson document is a

3 September 1961 memo from G.J. Brabb ("Brabb 1961") which set

4 forth a "quantitative speculation" that "stack losses getting

5 through the filters probably account for 1 to 2 kilograms per

6 year." (Klementiev March 1996 Rpt. at pp. 15-17); (Brabb Rpt. at

7 p. 5); (Klementiev Dep. at p. 449). Based on Anderson and Brabb,

8 Klementiev provided a release estimate of 1,168 Ci (18.68 kg) in

9 his March 1996 report. Klementiev's February 1997 report

10 modified this slightly, giving a mean figure of 1,122 Ci (17.95

11 kg) within a 95% certainty range of 947 Ci to 1,294 Ci.

12 (Klementiev 1997 Rpt. at App-12).

13   Defendants are not concerned about Klementiev's first two

14 estimates, claiming they are so small they cannot produce doses

15 exceeding the doubling doses from which causation can be

16 inferred. It is Klementiev's third estimate, and his largest

17 estimate, which is at the center of the controversy and

18 therefore, the focus of defendants' motion in limine. This

19 estimate- 20,000 Ci (approximately 320 kg)[336] - has the

20 potential for producing plutonium doses exceeding the doubling

21 doses for various types of cancer.

22   Klementiev's third estimate is based on an engineering

23 analysis or "process analysis" of various operations associated

24 with the plutonium finishing process. He identifies four PFP

25 ―――――――――――――――――

   [336] Estimate of total plutonium release for the time period
26 in question is mean value of 323 kg, with a 95% certainty or
   confidence range of 36.67 kg to 850 kg (2,271 Ci to 52,190 Ci).
27 (Klementiev 1997 Rpt. at pp. 17 and App-6).
28
   ORDER RE SUMMARY JUDGMENT-   429

1 processes and estimates the plutonium releases to the ventilation

2 system and eventually to the environment for each of the

3 processes. For each process he: (1) determines the total amount

4 of plutonium processed per year; (2) multiplies the amount by a

5 percentage he claims was released to the air during processing

6 (the ARF or "airborne release fraction"); (3) multiplies the

7 product of (1) and (2) by the percentage of airborne material he

8 claims would not have been removed from the air by the filtration

9 system (filter penetration fraction); and (4) multiplies this

10 result by the number of years each of these processes was part of

11 PFP operations.[337] The four processes examined by Klementiev

12 include 231-Z Casting; 231-Z Burning; 234-5Z Processing; and 234-

13 5Z Casting.

14   For 231-Z Casting, Klementiev posited the amount processed

15 per year was 2,360 kg of which 3% became airborne (70.8 kg) and

16 was drawn to the ventilation filters. He estimated the filters

17 removed all but 3.25% of the 70.8 kg, resulting in a yearly mean

18 release of 2.34 kg. Over a 23 year period (1955-77), the total

19 mean release was 54.9 kg. (Klementiev 1997 Rpt. at p. 16).

20   For 231-Z Burning, Klementiev posited the amount processed

21 per year was 590 kg of which 1.4585% became airborne and was

22 drawn to the ventilation filters. He estimated the filters

23 removed all but 3.25% of that amount, resulting in a yearly mean

24 release of 0.28 kg. Over a 19 year period (1959-77), the total

25 mean release was 5.3 kg. (Klementiev 1997 Rpt. at pp. 16 and

26 _____

[337] This description is taken from defendants' brief. The
27 plaintiffs do not take issue with this description.

28 **ORDER RE SUMMARY JUDGMENT-   430**

1   17).

2   For 234-5Z Processing, Klementiev posited the amount

3   processed per year was 4,602 kg of which 7% became airborne and

4   was drawn to the ventilation filters. He estimated the filters

5   removed all but 3.25% of that amount, resulting in a yearly mean

6   release of 10.45 kg. Over a 23 year period (1955-77), the total

7   mean release was 240 kg.   (Klementiev 1997 Rpt. at p. 17).

8   For 234-5Z Casting, Klementiev posited the amount processed

9   per year was 2,071 kg of which 3% became airborne and was drawn

10 to the ventilation filters. He estimated the filters removed all

11 but 3.25% of that amount, resulting in a yearly mean release of 2

12 kg. Over an 11 year period (1955-65), the total mean release was

13 22.4 kg.   (Klementiev 1997 Rpt. at p. 17).

14   Based on Klementiev's figures, the total annual release of

15 all four processes was 15.1 kg (15.07 kg) and the total

16 cumulative release was 323 kg (322.6 kg).   (Klementiev 1997 Rpt.

17 at p. 17).

18

19   **(a) Reliability**

20   According to defendants, a threshold problem is that

21 Klementiev provides three mutually exclusive estimates, but is

22 unwilling or unable to say which of the three he considers most

23 reliable. Indeed, at his deposition, Klementiev testified that

24 all three estimates "deserve to be considered seriously" and that

25 it was not a matter of him saying one estimate was "more trustful

26 than another." He referred to his third estimate (20,000 Ci or

27 320 kg) as the most "sophisticated" and "detailed" of his

28 **ORDER RE SUMMARY JUDGMENT-   431**

1 estimates since it "incorporates more details, more evidence,

2 more data, more observations." (Klementiev Dep. at p. 462).

3   Klementiev refused to assess his third estimate in terms of

4 reliability because he could not "measure this reliability."

5 Indeed, he stated he could not measure the reliability of any of

6 his three estimates. According to Klementiev, he did not have

7 the data necessary to measure reliability. (Id. at pp. 462 and

8 465). Klementiev also refused to opine which of his release

9 estimates he considered most "probable." (Id. at pp. 465-66).

10   Klementiev indicated he was not sure whether his three

11 estimates overlapped, but suggested they probably did. (Id. at

12 pp. 468-70). Defendants assert the estimates do not overlap and

13 if one of them is right, the other two must be wrong.

14   The plaintiffs say Klementiev considers all his calculations

15 to be reliable but he "does not intend to present 3 different

16 reports to a jury, just the last and 'more realistic.'"

17 Plaintiffs cite a portion of Klementiev's deposition where he

18 testified that the "finding" in his October 1996 report was "more

19 realistic" than his previous one (the March 1996 report), noting

20 he had "more information " and "more evidence." (Klementiev Dep.

21 at p. 527). In his October 1996 report, Klementiev first laid

22 out his "process analysis" which he then supplemented in his

23 February 1997 report.

24   Plaintiffs leave no doubt upon which estimate Klementiev

25 intends to rely. Therefore, the court need only consider the

26 reliability of his "last" and largest estimate- 20,000 curies or

27 320 kg.

28

ORDER RE SUMMARY JUDGMENT-   432

(i) Monitoring Data

Defendants contend Klementiev's 20,000 Ci estimate contradicts all the available empirical data. According to defendants, the large amount of plutonium Klementiev estimates was released to the environment is not consistent with the monitoring data, and he makes no effort to explain the inconsistency.

-- Stack Sampling Data

Klementiev's first estimate of cumulative plutonium releases- 0.15 Ci- is based on stack sampling data, more specifically air measurements taken from the Z-Plant stack (291-Z Stack). As noted above, Klementiev testified that all three of his estimates, including his first estimate, "deserve[d] to be considered seriously." The problem, say defendants, is that Klementiev does not explain how his release estimate of 20,000 curies is to be reconciled with his estimate of 0.15 curies.

Plaintiffs contend HEDR's analysis of plutonium releases from the **separations plants** was based on a limited set of stack measurements. In contrast, plaintiffs say Klementiev in his March 1996 report "had available his spreadsheet of 455 data entries of stack measurements from operating documents that had been completely overlooked by HEDR."[338] Klementiev's 0.15 Ci estimate for the **Z-Plant** is based on this stack sampling data which is referred to and contained in his March 1996 report.

---

[338] See Appendix A to Klementiev's March 1996 Report.

ORDER RE SUMMARY JUDGMENT-   433

1 (pp. 14-15; Table 13 at p. 19).

2 Nonetheless, plaintiffs suggest this stack data is

3 "unreliable, inadequate, or too sporadic," thus justifying

4 Klementiev's "process analysis" which produces his 20,000 curie

5 estimate. They say their expert, Dr. Robert Jervis, has reviewed

6 the stack data and found it to be unreliable.[339]

7 In his March 1996 report, "Reliability Assessment of Pu

8 Release Estimates at Hanford ('48-'80)," Jervis stated that

9 improper design of stack sampler lines and stack nozzles was

10 "considerable ranging from 3 to 30 in underestimating Pu

11 releases." (Jervis 1996 Rpt. at p. 11). The defendants assert

12 that even if this is true, it would at best increase Klementiev's

13 estimate to 4.5 curies (0.15 Ci x 30). However, it appears that

14 Jervis' "3 to 30" opinion pertains only to the stack monitoring

15 data from the **separations plants**, and not the **Z-Plant**.

16 Plaintiffs argue that in addition to Jervis' opinion being

17 confined to the **separations plants**, it does not take into account

18 other limitations in the stack monitoring data identified by him,

19 including "Errors of Thick-Sample Alpha Counting" and

20 "Inefficiency of Air-Sampling Filters." (Jervis 1996 Rpt. at pp.

21 10-11).

22 In his March 1996 report, Jervis dedicated a specific

23 section to "Pu Losses from the Z-Plant Stacks and MUF [Material

24 Unaccounted For]." Jervis referred to Brabb's conclusion that

25

_____

26 [339] Jervis is a Professor Emeritus in the Department of
Chemical Engineering and Applied Chemistry at the University of
27 Toronto.

28

ORDER RE SUMMARY JUDGMENT-   434

"stack losses getting through the filters probably account for 1 to 2 kilograms per year (of MUF)." According to Jervis, "[t]his quantity represents an appreciable amount of material unaccounted for (MUF) and exceeds by several orders of magnitude the few grams per year of total estimated Hanford stack discharges obtained from **monitoring**." (Jervis 1996 Rpt. at pp. 11-12)(Emphasis added). It appears, however, Jervis did **not** discuss purported **inadequacies** in the **Z-Plant** stack monitoring. Rather, he simply opined the monitoring data was not consistent with Brabb's inventory analysis.

In addition to Jervis' March 1996 report, plaintiffs refer to certain documents which they say relieve Klementiev of any obligation to rely upon stack monitoring data as the foundation for his Z-Plant plutonium source term estimate. These include a 1959 GE document referring to damaged filters (J.H. Palmer, "Condition of Commercial High Efficiency Filters Upon Arrival and/or Installation," July 1959, Foulds Ex. 233); an August 1961 document, "Sources of SS Material Losses and their Relation to B-PID[340] in the Chemical Processing Department," authored by a W.H. Johnson (Johnson 1961, Foulds Ex. 212); and an October 1959 report, "Control and Inspection Systems for Plutonium Production," authored by C.A. Bennett, et al. (Bennett 1959, Foulds Ex. 143). According to plaintiffs, the latter two documents (Johnson and Bennett, et al.) confirm the Z-Plant stack was an "unmonitored waste stream."

---

[340] Book-physical inventory difference.

ORDER RE SUMMARY JUDGMENT-   435

1   Plaintiffs quote the following passage from the Johnson

2 document:

3   CPD [Chemical Processing Department] has a
         record of numerous measurement and control
4   achievements. However, even though steady
         improvement in measurement capability has
5   been shown, there has not always followed
         a commensurate improvement in the material
6   balances. The advances made have been for
         the most part in measurement of product
7   streams, internal recycle, and inventory.
         Waste streams have received relatively little
8   **attention.**

9 (Johnson 1961 at p. 2)(Emphasis added).

10   Johnson, who was affiliated with "Nuclear Materials

11 Measurements, Nuclear Materials Operations," stated his report

12 was "intended to provide a starting point for a waste evaluation

13 program" and that the goal was "attainment of a certain level of

14 control or complete verification of all potential waste streams."

15 (Id.) In a section entitled "How **Can** Undetected Losses Occur,"

16 he listed, among other things, "[n]o measurement made on such

17 things as loss to atmosphere via stacks or unrecoverable

18 inventory in cell floors, etc." (Id. at p. 4)(Emphasis added).

19 He indicated that verification of wastes could include

20 "[i]nvestigation of presently unmeasured sources of loss such as:

21 Stack gas, buried waste, outside storage sumps, deposition on

22 cell floors, loss thru cracks in cell floors; hood filters."

23 (Id. at pp. 4-5). Under a section entitled "Where Can Losses

24 Occur in Places Not Now Monitored," Johnson listed "Plant off

25 gases." (Id. at p. 7). And in another section entitled "Most

26 **Likely** Causes and Sources of Undetected Losses," he listed "stack

27 losses." (Id.)(Emphasis added).

28
   ORDER RE SUMMARY JUDGMENT-   **436**

1   With regard to the Bennett document, plaintiffs refer to a

2   diagram of Z-Plant contained therein which they claim shows the

3   Z-Plant Stack was an "unmonitored stream." (Bennett 1959, Figure

4   4 at p. 44). As best as the court can make out, the diagram

5   states "light lines denote unmonitored streams." According to

6   defendants, "heavy lines" denote streams **not** "presently monitored

7   for **accountability** purposes." However, the court reads the

8   diagram to say that **"heavy lines denote streams presently**

9   **monitored for accountability purposes."** The court also cannot

10   tell for sure if there is a "heavy line" or a "light line"

11   running from the vent exhaust to the stack. The defendants

12   apparently assume it is a "heavy line" which therefore means the

13   vent exhaust to the stack was **not** monitored for **accountability**

14   purposes.

15   Defendants assert that in relying on the Johnson and Bennett

16   documents, the plaintiffs ignore the distinction between the

17   Hanford organizations responsible for **radiation monitoring** and

18   those responsible for **tracking nuclear materials inventory** (i.e.

19   **"accountability"**). It is clear from Johnson's report that he was

20   concerned with the inventory aspect (aka "material balances").

21   Bennett's report is concerned with how best to control the

22   inventory "to detect any diversion of nuclear materials in

23   sufficient quantity to increase a stockpile of nuclear weapons."

24   (Bennett 1959 at p. 3).

25   Furthermore, as is obvious from Klementiev's March 1996

26   report, some radiation monitoring (stack monitoring) undoubtedly

27
28   **ORDER RE SUMMARY JUDGMENT-   437**

occurred at the Z-Plant.[341]   Appendix A to Klementiev's March

1996 report contains his "Source Spreadsheet Data."   In it he

lists each of the documents relied upon for sampling data,

including data pertaining to the "Z-Plant," the "Z-Plant 291 Z-

Stack," "291-Z-1 Stack" and the "291-Z Building Stack."

Klementiev used that data to figure a yearly plutonium release

estimate from the Z-Plant commencing in 1950 and ending in 1969.

Table 13 of Klementiev's March 1996 Rpt. (at p. 19) shows those

yearly plutonium release estimates producing a cumulative

estimate of 0.15 Ci (based on historical records alone).[342]   In

Table 14 (p. 19), Klementiev doubles his yearly release estimates

and in turn, his cumulative value (to 0.30 Ci) "because of

accounting for missed submicron particles."[343]   In Table 16 (p.

20), Klementiev takes into account the Brabb memo in calculating

his yearly estimates.   This inventory analysis produces the 1,168

Ci figure, referred to previously as Klementiev's "second

estimate."[344]

In his March 1996 report, Klementiev referenced McConnon,

"The status of gaseous effluent monitoring at HAPO, December

1961," 1962 HW-69205, General Electric Company, Richland,

---

[341]   The court finds it interesting that at the same time
plaintiffs tout their ability to uncover additional stack
monitoring data and Klementiev's reliance on this additional
data, they quickly dismiss it as "inadequate."

[342]   Klementiev refers to this as "Scenario C."

[343]   Klementiev refers to this as "Scenario D."

[344]   Klementiev refers to this as "Scenario F."

ORDER RE SUMMARY JUDGMENT-   438

Washington (hereinafter, "McConnon 1962").[345] McConnon's report

was intended as "a comprehensive survey of the methods used for

sampling radioactive materials emitted from plant stacks and

compilation of the results from the various sampling programs for

1961." (McConnon 1962 at p. 1). In his survey, McConnon

indicated:

> CPD RM [Chemical Processing Department Radiation
> Monitoring] collect a **daily sample** of the exhaust
> gases from the **291-Z Stack**. A vacuum steam jet
> draws the 5.0 CFM sample through a 4" x 4" H-70
> filter. After a 24-hour period to allow for decay
> of natural air-borne alpha emitters, the sample is
> counted with an alpha scintillation detector. Any
> activity found is assumed to result from plutonium.

(Id. at p. 9)(Emphasis added). McConnon provided a table showing

the daily average and maximum emissions (in microcuries) for all

months of 1961.[346] (Id. at p. 27, Table D).

Defendants also note that Postma, et al., "Radioactive

Particles In the 234-5 Building Ventilation Exhaust" (1959),

provides a description of the sampling system used at the Z-

Plant. (Postma, et al., 1959 at pp. 3-4; Foulds Ex. 239). The

document states "[t]he 234-5 Building ventilation exhaust is

continuously sampled for the purpose of estimating the amount of

radioactive (alpha emitting) material discharged to the

atmosphere." (Id. at p. 3)(Emphasis added).

---

[345] Defendants' Ex. 212.

[346] 1961 is significant because this is when Brabb's memo
was prepared reporting the inventory shortage and making the
"quantitative speculation" that 1 to 2 kg a year was going out
the stack. In Table 13 of his March 1996 report, Klementiev
provides a release estimate of 0.01204 curies (0.000196 kg) for
1961 (Table 13 at p. 19), which is very small compared to Brabb's
estimate of 1 to 2 kg.

ORDER RE SUMMARY JUDGMENT-   439

1   In their brief, plaintiffs acknowledge McConnon 1962, but

2 claim it represents the **"first** formal, tabulated report of Z

3 Plant emissions . . . ." (Plaintiffs' Response Br. at pp. 52-

4 53)(Emphasis in text). Plaintiffs say that "[f]rom the time a

5 substantial MUF (material unaccounted for) began to accrue at Z

6 Plant, one might have reasonably expected the contractor to

7 include Z Plant stack emissions in the periodic environmental

8 reports," and that "[i]t is shocking to review quarterly and

9 annual reports for 1956 through 1960 to see 200-foot stack

10 releases for each of the 100 Area Reactors, for Redox, Purex and

11 the UO3 Plants in the 200 Area, but not a single mention of the Z

12 Plant."

13   Plaintiffs also acknowledge Postma 1959, but argue that it

14 raises issues about losses from the sampling line to the Z Plant

15 stack plenum. One of the conclusions reached by Postma was that

16 "[t]he present sampler could not be relied on to representatively

17 sample large [plutonium] particles." (Postma 1959 at p. 10).

18 Plaintiffs cite several other documents which they claim show

19 there were flaws in the Z-Plant stack sampler. (Plaintiffs'

20 Response Brief at pp. 56-57). Plaintiffs go on to cite their

21 expert, Dr. Jervis, for the proposition that "particles not lost

22 to the sampling line, but which reach the collector may still

23 penetrate the filter paper and pass uncounted." They say Jervis

24 opines that the filter paper used for sampling is transparent for

25 submicron particles. (_Id_. at p. 57).

26   At his deposition, Klementiev acknowledged that stack

27 sampling and filter efficiency was not his area of expertise and

28

ORDER RE SUMMARY JUDGMENT-   440

1 that he had not been responsible for analyzing those particular

2 topics. (Klementiev Dep. at pp. 545-49). In his reports,

3 Klementiev relied on Dr. Jervis (Jervis' 1996 report) for

4 assessment of stack sampling and filter efficiency. He did not

5 independently analyze those issues, nor apparently could he based

6 on his lack of expertise. Plaintiffs acknowledge Jervis is their

7 "monitoring expert." Indeed, they claim that in the absence of

8 any challenge to Dr. Jervis, they are not obligated to answer

9 defendants' monitoring claims, but do so only out of an

10 "abundance of caution." Plaintiffs say Klementiev's task was

11 solely "source term." (Plaintiffs' Response Br. at p. 49).[347]

12   Jervis submitted a supplemental report in March 1997 which

13 was stricken by the court on the basis that it did not meet the

14 supplementation criteria. (July 14, 1997 Order at Ct. Rec. 987).

15 Plaintiffs allude to this in their brief:

> . . . Jervis' educational and professional
> background and training in radiation monitoring
> qualifies him to evaluate the magnitude of
> Klementiev's estimates which he did in his
> supplemental report of 1997. Since this report
> was stricken by the Court, the plaintiffs will
> not reference the substance of Professor Jervis'
> opinions in this section. However, the analysis
> on MUF issues addressed in Jervis' 1997 report
> rebuts the implication put forth in defendants'
> motion that Klementiev's analysis was performed
> in a vacuum. Further, as Klementiev testified,
> he was in communication with Dr. Jervis and had
> established [an] ongoing consensus that in
> Klementiev's mind informally sufficed for a formal
> explicit approval.

_____

[347] According to plaintiffs: "Jervis is plaintiffs' expert
on filter efficiencies and sampling, he was not retained for the
purpose of calculating a source term- that task was left to
Klementiev." (Response Brief at p. 21).

ORDER RE SUMMARY JUDGMENT-   441

1   (Plaintiffs' Response Br. at p. 21).

2   The court need not pay any heed to Jervis' 1997 report, but

3   since the plaintiffs have brought it up, the court accepts their

4   invitation to determine whether Klementiev's analysis was

5   "performed in a vacuum."   In his 1997 report, Jervis indicated

6   his March 1996 opinion that Pu releases were underestimated by a

7   factor of 3 to 30 actually took into account **all** of the

8   limitations and errors he identified as pertaining to stack

9   monitoring: 1) errors of thick sample alpha counting; 2)

10   inefficiency of air-sampling filters; **and** 3) improper design of

11   stack sampler lines and nozzle.   (Jervis 1997 Rpt. at p. 13,

12   Section 6.1).   Jervis stated:

13   Hanford stack monitoring, both at Chemical
             Processing[348] and the Z-Plant Site was grossly
14   underestimating Pu releases by what now appears
             with the aid of new information, by a factor of
15   up to 500, so that instead of reported aggregate
             releases of tens of grams per year, actual releases
16   amounted to kilograms/year, i.e. in the range inferred
             on other bases and, those calculated by Klementiev
17   [Kl96]. . .

18   (Jervis 1997 Rpt. at p. 14, Section 6.3).

19   Jervis' reference to "Kl96" is Klementiev's October 1996

20   report.   (Jervis 1997 Rpt. at p. 17, "References").   In that

21   report, Klementiev's range is between 0.08 kg/yr and 3.5 kg/yr

22   for "minimum conditions" and 23.8 kg/yr for "more realistic

23   conditions."   (Klementiev October 1996 Rpt. at p. 18).

24   Jervis' 1997 analysis took into account the Palmer and

25   Johnson documents (Palmer 1959; and Johnson 1961) which, as

26   _____

27   [348]   Separations Plants

28   **ORDER RE SUMMARY JUDGMENT-**   **442**

mentioned above, plaintiffs contend relieved Klementiev of any

obligation to rely upon stack monitoring data as the foundation

for his Z-Plant source term estimate.

Elsewhere in his 1997 report, Jervis stated:

> Klementiev has made new estimates based on some
> information recently made available and several
> scenarios considered indicate that average
> annual Pu emissions were probably . . . of the
> order of 2-40 kg/y [K196] with 95% uncertainty
> limits [K197].[349] Releases of this magnitude
> are comparable in magnitude to MUF estimates,
> made during peak years of operation, of 75-125
> kg/y, **but there were other probable sources of
> MUF in addition to losses through the stacks.**

(Jervis 1997 Rpt. at p. 10)(Emphasis added).

Defendants point out a couple of very pertinent things about

Jervis' 1997 report: 1) even assuming Jervis was correct in

stating that the stack monitoring data is off by a factor of up

to 500, this would only increase Klementiev's 0.15 Ci estimate to

75 Ci (0.15 x 500)[350], still a far cry from his 20,000 Ci

estimate; and 2) Jervis did not specifically endorse any of

Klementiev's estimates, and in particular the 20,000 Ci

estimate.[351]

_____

[349] Klementiev's February 1997 report.

[350] 75 Ci is equal to 1.2 kg (75 x 0.016).

[351] In his March 1996 report, Jervis opined that "PuO2 in
finely divided form was undoubtedly by-passing and penetrating
the stack ventilation systems in appreciable quantities and led
investigators to the conclusion that (in 1961) as much as 1 to 2
kilograms per year were probably lost to the environment and
contributed to an estimated 40-50 k of unaccounted losses [those]
years." (Jervis 1996 Rpt. at p. 14). In his March 1997 report,
he opined that "[t]he public health and environmental
consequences of releasing about 50 kg of submicron, dispersible
and highly respirable plutonium at Hanford are considerable."
(Jervis 1997 Rpt. at p. 15).

ORDER RE SUMMARY JUDGMENT-   443

1   Jervis spoke only in the most general terms of actual

2   releases being in the range calculated by Klementiev. One of the

3   figures within that range is 0.08 kg a year which over a 25 year

4   period would amount to a total of only 2 kg. Furthermore, Jervis

5   was careful to say that even though Klementiev's release

6   estimates were comparable in magnitude to MUF estimates, **"there**

7   **were other probable sources of MUF in addition to losses through**

8   **the stacks."**[352]

9   _____

10   This court struck Jervis' 1997 report on the basis that it
did not materially alter the analysis contained in his 1996
report, but simply bolstered and restated the conclusion of his
11   1996 report- approximately 50 kg released. 50 kg is equivalent
to approximately 3,125 curies (50 kg/0.016), which is still six
12   times less than Klementiev's 20,000 Ci (320 kg) estimate.
     Note also that Jervis' 50 kg estimate is based on Brabb's
13   inventory analysis. It assumes the accuracy of Brabb's
"quantitative speculation" of a 1 to 2 kg annual release of
14   plutonium via the stack over a period of 25 years (1950-1975).
(Jervis 1997 Rpt. at p. 14). The 50 kg estimate is not the
15   result of Jervis applying a 500 factor adjustment to any
particular figure. The 500 factor pertains only to the stack
16   monitoring data. Accordingly, it is appropriate for defendants
to apply the 500 factor to Klementiev's 0.15 Ci estimate which is
17   based on stack monitoring data.

18   [352] At his deposition, Klementiev acknowledged that other
factors could account for MUF including unrecorded disposal of
19   plutonium to waste, excess plutonium conversions, unrecorded
burials of plutonium on equipment, and limitations in sampling
20   and measurement of liquids and solids. (Klementiev Dep. at pp.
754-55). As noted above, Bennett, et al., 1959, was concerned
21   about the potential theft of plutonium.
     In their brief, plaintiffs discuss the Z-9 Crib.
22   (Plaintiffs' Response Br. at pp. 61-62). The Z-9 Crib was a
buried storage tank in which assayed liquid waste materials from
23   the Z-Plant were discarded. Plaintiffs cite Anderson 1977 which
indicates that when the crib was mined in 1973, 100 kg of
24   plutonium was discovered, 73 more kilograms than the assigned
book value of 27 kg. (Anderson 1977 at p. 4). Plaintiffs assert
25   this shows that historical monitoring results of Hanford waste
streams, whether liquid or airborne discharges, cannot be trusted
26   for dose reconstruction purposes. On the other hand, it also
supports the notion that factors, other than stack release, could
27   account for MUF. Indeed, as defendants point out, Anderson did

28   **ORDER RE SUMMARY JUDGMENT-   444**

1   In sum, plaintiffs cannot so easily dismiss the stack

2   monitoring data. Plaintiffs say Klementiev does not have to rely

3   on that data because of its shortcomings, but Jervis analyzed

4   those shortcomings and in doing so, relied upon all of the

5   pertinent documents cited by plaintiffs. Even assuming the truth

6   of Jervis' findings, it does not support a 20,000 curie estimate.

7   As noted, Klementiev was not willing to just throw his 0.15 Ci

8   estimate on the trash heap, saying it deserved serious

9   consideration.

10

11   **-- Air Concentration Data**

12   Hanford has published measurements of plutonium in the air

13   in Richland over three decades.[353] According to defendants, the

14   measurements show the same level of plutonium in the air as at

15   other locations around the U.S., indicating the plutonium air

16   concentrations at Richland were the result of U.S. and Soviet

_____

18   not attribute any of the MUF to stack releases. The
     "possibilities" identified by Anderson included: 1) equipment
19   burial transfers; 2) vacuum system depositions; 3) liquid
     discharge transfers; 4) scrap receipts; and 5) routine burials.
20   (The "MUF" issue is discussed _infra_).

21   [353] Defendants indicate three studies have published
     Richland air data, including: Perkins, R.W., et al.,
22   "Measurements of Airborne Radionuclides and Determination of
     Their Physical Characteristics," in Klement, A.W., ed.,
23   **Proceedings of the Second Conference, AEC, Division of Biology
     and Medicine, Germantown, Maryland** (Nov. 1965) (Defendants' Ex.
24   180); Perkins, R.W., and Thomas, C.W., "Worldwide Fallout," in
     Hanson, W.C., ed., **Transuranic Elements in the Environment**
25   (DOE/TIC-22800) (1980) (Defendants' Ex. 181); Pan V. and
     Stevenson, K.A., "Temporal Variation Analysis of Plutonium
26   Baseline Concentration in Surface Air from Selected Sites in the
     Continental US," 32 **J. Envir. Radioactivity** 239 (1996)
27   (Defendants' Ex. 179).
28
     ORDER RE SUMMARY JUDGMENT-   445

weapons tests conducted at the time of the measurements.   Even

assuming the measured air concentrations were due to Hanford

releases, defendants say the concentration levels are thousands

of times less than what they would be if Klementiev's release

estimates were correct.

Defendants cite deposition testimony from Klementiev

indicating he was not familiar with and had not reviewed this air

concentration data.   (Klementiev Dep. at pp. 556-58); that he did

not review the air concentrations Dr. Stewart calculated based on

Klementiev's estimates (Id. at pp. 437-38); that he did not know

if anyone had compared the plutonium air concentrations measured

around Hanford with the air concentrations produced by Stewart's

model (Id. at p. 561); and did not have any evidence that the

level of plutonium in the air around Hanford was greater than the

average levels measured in other locations around the United

States.   (Id. at p. 573).

The plaintiffs assert "Dr. Klementiev is not alone in

rejecting the environmental monitoring data for use in dose

reconstruction."   They cite Hanf, et al., "Environmental

Radiological Monitoring of Air, Rain, and Snow on or near the

Hanford Site, 1945-1957" (March 1994), a HEDR document[354], which

reported that during the 1940s and early 1950s the equipment and

techniques used for collecting radiological samples were often

inaccurate.   Accordingly, "[t]he result was the air monitoring

data are insufficient for use in the HEDR project."   (Hanf, et

_____

[354]   PNWD-2234-HEDR.   (Foulds Ex. 188).

ORDER RE SUMMARY JUDGMENT-   446

1   al., 1994 at p. 1).[355]

2   Plaintiffs also point out what they say are a number of

3   limitations to drawing inferences from the studies cited by

4   defendants (i.e. inadequate filters for the purpose of collecting

5   samples). However, in doing so, there is no citation to any

6   expert reports. Klementiev did not analyze the air concentration

7   data. Plaintiffs do not cite Jervis for any specific points

8   relating to air concentration data. There is no indication that

9   he critiqued such data or tried to reconcile it with Klementiev's

10   release estimates.[356]

11   Plaintiffs apparently expect the court to rely on their

12   counsel for an opinion as to whether or not air concentration

13   data should have been consulted in formulating release estimates.

14   This is inappropriate. There may be perfectly valid reasons for

15   disregarding monitoring data, but the point is that the reasons

16   need to be supplied by an expert. They cannot just be presumed,

---

[355] The studies cited by defendants appear to have measured
air concentration for periods **after** 1957. (Defendants' Exs. 179,
180 and 181). That may be of importance considering a
significant amount of PFP activity occurred prior to 1957.
Defendants do not say if HEDR's estimate of 1.78 Ci from the
separations plant was compared to any air concentration data.
However, even if HEDR did not make such a comparison, that does
not necessarily mean Klementiev or plaintiffs' experts acted
scientifically in disregarding air concentration data.

[356] It appears Jervis limited himself to stack sampling
issues, including efficiency of filters used as part of the Z-
Plant stack sampling system, not those used to take environmental
samples. Plaintiffs appear to argue in their brief that the Z-
Plant filters were similar to those used to take environmental
samples and therefore, should be considered inadequate. Once
again, however, plaintiffs do not indicate which of their experts
may have arrived at such a conclusion and what basis they have to
support it. Efficiency of the Z-Plant filters is discussed
infra.

ORDER RE SUMMARY JUDGMENT-   447

1 nor can counsel fill the gaps.

2

3   -- **Soil Sampling**

4   At his deposition, Klementiev acknowledged he had not

5 reviewed any measurements showing the concentration of plutonium

6 in the soil around Hanford (Klementiev Dep. at p. 574); did not

7 compare those historical measurements with the soil-deposition

8 concentration that would be predicted based on his release

9 estimates and Dr. Stewart's dispersion model (Id. at 525-26); and

10 had not verified if the levels of plutonium in the soil near

11 Hanford have ever been higher than the average at other locations

12 in the U.S. (Id. at p. 576).

13   According to defendants, if there were significant emissions

14 of plutonium, it would have deposited on the soil around Hanford.

15 Citing Price, K.R., "A Review of Historical Data on the

16 Radionuclide Content of Soil Samples Collected from the Hanford

17 Site and Vicinity," (PNL-6734)(Nov. 1988)[357], defendants assert

18 that routine measurements of soil have not yielded any evidence

19 of plutonium at a higher level than would be expected from

20 weapons testing fallout. Price summarized plutonium measurements

21 that had been reported in Hanford annual environmental monitoring

22 reports since 1971. According to Price, measurement of

23 radioactive materials in soil samples collected from onsite

24 (excluding operating areas) and offsite locations has been a

25 routine part of sitewide environmental monitoring at Hanford

26 _____

27   [357] Defendants' Ex. 182.

28 **ORDER RE SUMMARY JUDGMENT-   448**

since 1971.   (Price 1988 at p. 3).   Price also examined the

results of soil sampling contained in "special purpose studies."

It appears that even in the "special purpose studies" analysis of

soil for plutonium content did not take place until 1970.   (Id.

at pp. 5-11). Among Price's "observations" was that "[s]oil

sampling has not revealed any gross contamination of the offsite

environs from past Hanford operations."   (Id. at p. v,

"Summary").

The plaintiffs question the relevancy of the Hanford soil

monitoring, as well as its reliability.   They point out that

routine monitoring did not occur until 1971, although plutonium

production had been occurring for years prior to that, including

the period (early 60s') when Brabb found his inventory

discrepancy.   Plaintiffs' counsel cite a study, Hakonson, et al.,

"Ecological Relationships of Plutonium in Southwest Ecosystems,"

(1980)[358], which evaluated plutonium concentration in the soil

around the Trinity (New Mexico) Atomic Bomb Site.   According to

counsel, plutonium particles dissipate over time by

"resuspension" transport mechanisms, such that soil sampling in

subsequent years will not measure the true extent of deposition

from previous years, even if the sampling was conducted

properly.[359]   Plaintiffs note the Trinity results show

_____

[358]   Foulds Ex. 187.

[359]   "Resuspension" means that the particles will not
necessarily remain in the soil.   Essentially, they can be blown
about to new locations (i.e. become "resuspended" in the
atmosphere).   This is significant since Pu has a very long period
of radioactive decay.

ORDER RE SUMMARY JUDGMENT-   449

1 decreasing concentrations of plutonium in the soil from 1950 to

2 1973. (Id. at p. 409, Table 3). Thus, say plaintiffs, the

3 sampling of soil around Hanford from 1971 onward would not

4 accurately reflect the plutonium emitted from the Hanford plant.

5   This "resuspension" argument may have some validity.

6 Defendants do not challenge the theory itself. However, the

7 problem again is plaintiffs do not indicate that any of their

8 experts opine this is a valid basis for disregarding Hanford soil

9 monitoring data. Klementiev made it clear that his job was only

10 source term, and specifically stack releases. (Klementiev Dep.

11 at p. 524). Plaintiffs do not give any indication that Jervis,

12 or even more importantly, Stewart, offered any analysis of

13 Hanford soil monitoring data and why it should be disregarded on

14 the basis of the "resuspension" theory or because it did not

15 measure submicron particles.

16   According to plaintiffs, by 1971, laboratory analysis of

17 environmental samples was being conducted by U.S. Testing

18 Laboratory. For this reason, plaintiffs take issue with the

19 reliability of the Hanford soil monitoring data. They note, (as

20 they did in their river submission), that the EPA suspended its

21 contract with U.S. Testing; U.S. Testing pled guilty to fraud in

22 New Jersey; and Batelle subsequently conducted its own

23 investigation of U.S. Testing and in 1990 terminated its contract

24 with U.S. Testing. They also note that in Price 1988, the author

25 indicated that a "rigorous evaluation of [the soil monitoring]

26 data . . . was not conducted." (Price 1988 at p. v). Plaintiffs

27 essentially argue that because of this, their experts were not

28 **ORDER RE SUMMARY JUDGMENT-   450**

1   obligated to review any of the Hanford soil monitoring data.

2   There may indeed be questions about the validity of this

3   data for the reasons cited by counsel. However, the court is not

4   persuaded that gives plaintiffs' experts license not to make some

5   inquiry about, or analysis of, the historical measurements as

6   part of a methodologically sound scientific investigation.

7   Indeed, it seems likely that any flaws in the data due to

8   improper collection procedures, etc., would be revealed by such

9   an investigation.

10

11   -- **Autopsy Data**

12   According to defendants, the published scientific literature

13 contains measurements showing the levels of plutonium in the

14 tissue of persons living near Hanford are no different than the

15 levels in persons living in other locations in the U.S.

16   Defendants cite Nelson, I.C., et al., "Plutonium in Autopsy

17 Tissue Samples," 22 **Health Physics** 925 (June 1972)[360], which

18 concluded "[t]he reported measurements of plutonium in tissue

19 samples obtained at autopsy from former Hanford employees and

20 residents in the plant environs continue to demonstrate that,

21 while for some workers measurable plutonium does occur in the

22 body, the majority of the workers and residents have not received

23 significantly measurable amounts of plutonium." (Id. at 929).

24   Defendants cite another article from Nelson, "Plutonium in

25 South-Central Washington State Autopsy Tissue Samples- 1970-75,"

26 _____

27   [360] Defendants' Ex. 177.

28 **ORDER RE SUMMARY JUDGMENT-**   451

65 **Health Physics** 42 (Oct. 1993) (Defendants' Ex. 178) which

found:

> Although the number of cases is small, and
> the results are often very close to the limits
> of detection, the data indicate that during
> the period 1970-75, the majority of Hanford-
> site workers and nearby residents coming to
> autopsy had tissue concentrations of plutonium
> no larger than persons who lived farther away
> from Hanford and whose likely source of
> plutonium was limited to nuclear weapons
> testing fallout. Thus, on this basis, Hanford
> operations had made no significant addition to
> the occurrence of plutonium in people at the Hanford
> environs during the period in which the individuals
> sampled postmortem were alive.

(_Id_. at p. 425).

Klementiev acknowledged he did not review any of this

autopsy tissue data (Klementiev Dep. at p. 581), and did not have

any information that tissue data from Hanford showed levels of

plutonium higher than that measured elsewhere in the U.S. (_Id_. at

p. 583). Indeed, Klementiev testified it was beyond the scope of

his research to analyze autopsy tissue data. (_Id_. at p. 581).

Defendants say it was unscientific for Klementiev to not make any

attempt to reconcile such data with his own release estimates.

The plaintiffs assert the autopsy data cannot be used for

validation purposes because the studies are "few and are subject

to serious deficiencies." According to plaintiffs, there are

"enormous technical difficulties," one of which is that "the

alpha particle emitted cannot penetrate even a sheet of paper,

making its detection very difficult and requiring a chemical

separation of plutonium from the mass of tissue in which it was

embedded." They cite Jervis' 1996 report for that proposition

ORDER RE SUMMARY JUDGMENT-   452

1 (Jervis 1996 Rpt. at p. 10, Section 2.3.1). However, while

2 Jervis discusses "Errors of Thick-sample Alpha Counting" in the

3 context of stack monitoring, he does not discuss anything about

4 autopsy tissue data.

5   Plaintiffs also cite a 1977 paper from Nelson, "Plutonium in

6 Human Lung in the Hanford Environs," BNWL-SA-5855, which

7 describes the conversion from autoradiography measurement to

8 alpha spectrometry and states the use of the new procedure may

9 suggest "systemic error" in earlier measurements. Here again,

10 however, plaintiffs do not indicate that any of **their experts, in**

11 **their reports**, cited this as a scientifically valid basis for

12 disregarding autopsy tissue data.

13   The plaintiffs get a little bit closer to resolving that

14 problem when they cite deposition testimony from their expert,

15 Dr. Crawford-Brown, explaining why he did not use autopsy data to

16 determine plutonium doses to bone surfaces in particular

17 individuals. (Defendants' Response Br. at pp. 67-68). Crawford-

18 Brown testified that while autopsy data has begun to be useful in

19 estimating doses at low levels in tissues, there are problems in

20 measuring plutonium because of "difficulties in inter-individual

21 deposition patterns of the plutonium in the bone and therefore,

22 difficulties associated with selecting the right depth in tissue

23 in the bone surface to draw . . . samples from in determining

24 concentration." (Crawford-Brown Dep. at p. 261). There is no

25 indication from plaintiffs, however, that this was part of

26 Crawford-Brown's expert report(s). An expert is limited to the

27 opinions contained in his expert report pursuant to Fed. R. Civ.

28

ORDER RE SUMMARY JUDGMENT-   **453**

1 P. 26(a)(1)(B).

2 Finally, plaintiffs' counsel asserts the majority of

3 plutonium particles emitted were below the size level of

4 detection described in the autopsy studies cited by defendants.

5 Counsel cite a November 1997 memo from Jervis regarding particle

6 size (Foulds Ex. 310), but there is no indication that any of

7 plaintiffs' experts opined in their various reports that

8 plutonium particles would, for this specific reason, have escaped

9 detection during autopsy.[361]

10

11 **(ii) Klementiev's Airborne Release Fractions (Component of**

12 **Process Analysis)**

13 **-- 234-5Z Processing**

14 For the 234-5Z chemical processing operations, Klementiev

15 used an airborne release fraction (ARF) of 7% or 70,000 parts per

16 million (ppm). This figure is applied to the amount of plutonium

17 processed yearly (4,602 kg) to determine the amount of plutonium

18 lost to the PFP ventilation system. Beyond the ventilation

19 system are the PFP filters. Klementiev's analysis is that 3.25%

20 _____

21 [361] Defendants suggest plaintiffs should have attempted to
confirm Klementiev's release estimates by sampling urine in the

22 off-site population, due to the fact inhaled plutonium is
excreted very slowly. Defendants note that Klementiev was not

23 aware of any evidence that levels of plutonium in the urine of
members of the Hanford population have been higher than average

24 levels measured in the U.S. (Klementiev Dep. at p. 583).
The defendants do not cite to any published studies

25 regarding measurements of plutonium in human urine. It is one
thing to say plaintiffs' scientists are obligated to examine

26 existing scientific studies. It is quite another to say they are
obligated to undertake their own sampling project, especially if

27 no one else has previously done so.

28

ORDER RE SUMMARY JUDGMENT-   454

1 of the particles lost to the ventilation system penetrated the

2 filters and were released through the stack. For 234-5Z

3 Processing, the equation works this way: 4,602 x 7% x 3.25% =

4 10.47 kg released per year x 23 years = 240.9 kg total release.

5 This 241 kg represents nearly 75% of Klementiev's 320 kg total

6 release estimate (20,000 curies) for all of the plutonium

7 processes analyzed by him: 231-Z casting, 231-Z burning, 234-5Z

8 processing, 234-5Z casting.

9   According to defendants, the sole source for Klementiev's 7%

10 (70,000 ppm) ARF value is a 1968 paper by Jofu Mishima which

11 reports on **experiments** attempting to measure the ARFs for heated

12 plutonium powders. Mishima, J., "Plutonium Release Studies.

13 III. Release from Heated Plutonium Bearing Powders," (BNWL-

14 786)(July 1968) (Defendants' Ex. 176). Mishima concluded the

15 release fractions for "partially oxidized plutonium oxalate"

16 ranged from 570 to 8,200 ppm, much less than Klementiev's 70,000

17 ppm. (Id. at p. 5; Mishima Affidavit, Defendants' Ex. 174 at p.

18 4).

19   Defendants say Klementiev derived his 70,000 ppm value by

20 misinterpreting the following quote from Mishima's paper:

21   Combining the filter and deposition values
             does not alter the release rate significantly.
22   Assuming contamination did not significantly
             affect the chimney deposition rates, filter and
23   deposition values average approximately **7%**
             of the material carried through the chimney.
24
(Mishima 1968 at p. 25)(Emphasis added).
25
         In his affidavit, Mishima describes his experiment as
26
involving the placement of plutonium powder at the bottom of a
27

28 **ORDER RE SUMMARY JUDGMENT-   455**

1   quartz cylinder or chimney which was ventilated through a

2   membrane filter. The powders were heated to determine the

3   quantity of releases from heated plutonium powders. Mishima says

4   his experiment included measurements of plutonium deposited on

5   the **filter** and also on a **shim lining inside the chimney.**

6   (Mishima Affidavit, Paragraphs 4 and 5 at p. 2). According to

7   Mishima, the quote utilized by Klementiev describes the

8   measurements on the shim inside the chimney and explains that the

9   measurements on the filter were much higher. Mishima says it

10 also explains the shim measurements (deposition rates) were about

11 7% **of** the filter measurements. (<u>Id</u>. at Paragraphs 8 and 9, p. 3)

12 (Emphasis added). Simply put, Mishima asserts the 7% is not an

13 ARF value.

14   Table VI of Mishima 1968 shows the release rate for

15 particles carried through the chimney and collected on the filter

16 at 0.82 wt%/hr at 1000 degrees celsius, with air velocity through

17 the chimney of 100 cm/sec. For particles entrained but deposited

18 on chimney walls (on a shimstock liner), Mishima reported a

19 release rate of 0.057 wt%/hr. (Mishima 1968 at p. 22). This, of

20 course, comports with Mishima's conclusion that the release

21 fractions for "partially oxidized plutonium oxalate" ranged from

22 570 (0.057) to 8,200 (0.82) ppm. 570 is approximately 7% of

23 8,200.

24   Plaintiffs do not respond to defendants' assertion that

25 Klementiev misinterpreted the quote located at p. 25 of Mishima

26 1968, although they admit he relied upon the quote. Plaintiffs

27 assert that Klementiev additionally relied upon "the Table II

28 **ORDER RE SUMMARY JUDGMENT-   456**

1 experiments of Mishima reported in the same report, which have an

2 airflow that was flowing across and impinging somewhat upon the

3 sample of powder." According to plaintiffs, "Table II contains

4 the only such data similar to actual operational conditions at

5 Hanford, but these results were discarded by Mishima who felt

6 they were too high." (Plaintiffs' Response Br. at pp. 16-17).

7 Plaintiffs say Klementiev reviewed operational documents which

8 led him to rely on Mishima's Table II.[362]

9   Interestingly enough, however, the plaintiffs never cite

10 where in any of his reports Klementiev refers to Mishima's Table

11 II and explains why he would have done so (i.e. because it

12 purportedly simulated actual operating conditions). Defendants

13 note that in his October 1996 report, Klementiev specifically and

14 solely pointed to the quote at "Mi68, p. 25" as support for his

15 conclusion that the "average yearly filter load" was 7% for the

16 purpose of determining how much plutonium was released from PFP

17 chemical processing operations. (Klementiev October 1996 Report

18

19

20

_____

[362] Mishima says that he initially attempted to draw air directly into the bell-shaped section of the chimney through a side arm. According to Mishima, "[t]his arrangement caused the incoming air to jet upon the powder and produce high release rates" as set forth in his Table II. Table II indicates that with a contaminated air supply, at 1000 degrees celsius, and an air velocity of 100 cm/sec, the release rate was **7.7** (wt%/hr). (Mishima 1968 at p. 7)(Emphasis added).
    Mishima, however, did not choose this experimental method. He chose another method where the powder samples were placed on a stainless steel cap with air drawn "up and around" the cap through a space between the cap and the bottom of the chimney. (Id. at p. 9). This method produced the release rates set forth in Table VI (570 to 8,200 ppm).

ORDER RE SUMMARY JUDGMENT–   457

1   at p. 15).[363] As noted above, while Klementiev interprets the

2   7% as relating to filter load, Mishima makes clear he meant the

3   amount on the chimney lining was 7% (0.057) **of** the filter load

4   (0.82).

5   Defendants refer to portions of Klementiev's deposition

6   where he makes it quite clear that the quote on page 25 of

7   Mishima 1968 was the sole source of his 7% figure. For example,

8   Klementiev testified "I concentrated my attention on his

9   [Mishima's] report on the one part which is equivalent to

10   measuring before and measuring after [and] this part is on page

11   25." (Klementiev Dep. at p. 734). He reiterated that he

12   "focused on only one statement, which is located on page 25"

13   (Id. at p. 735), and once again that he was "actually focused on

14   the quote on page 25 . . . and as soon as I got that . . . direct

15   measurement, therefore I was satisfied with that measurement."

16   (Id. at p. 740).

17   Plaintiffs cite to portions of Klementiev's deposition

18   (pages 599 and 601) as purportedly showing that he relied upon

19   Table II found at p. 7 of Mishima. However, these citations

20   simply do not reveal such reliance. At p. 601 of his deposition,

21   Klementiev testified that "7 percent was not calculated by me,"

22   but was "given to me." There is no specific mention of Table II

23   and the 7.7% figure. At p. 599, Klementiev merely affirms that

24   _____

25   [363] In his February 1997 report, Klementiev stated only that
     "[t]he sources pertaining to plutonium-containing powders
     entrainment as the result of chemical processing were reviewed"
26   with the "likeliest" entrainment rate value being 7%.
     (Klementiev February 1997 Report at p. 13). He did not
27   specifically identify or discuss any of his sources.

28   **ORDER RE SUMMARY JUDGMENT-   458**

1   7% is the plutonium entrainment rate for chemical processing at

2   the PFP. Again, there is no mention of Table II and the 7.7%

3   figure. This is further confirmation that the 7% came from only

4   one place, page 25 of Mishima's report.

5   At his deposition, Klementiev was asked point blank by

6   plaintiffs' counsel whether he had relied on Table II in

7   calculating his 7% entrainment percentage for powder operations

8   at Hanford. Klementiev again made clear he had not calculated

9   the 7%, but took it from "historical records," specifically "Page

10 25" of Mishima 1968. (Klementiev Dep. at p. 846). Klementiev

11 added "I think that the reference to 7.7% percent is even more

12 harder evidence about the entrainment rate." (Id.). However,

13 plaintiffs have not cited to any portion of Klementiev's reports

14 or his deposition wherein he explains why the 7.7% is "harder"

15 evidence about the entrainment rate, or why the experiment

16 conducted by Mishima which produced the Table II results is more

17 representative of operating conditions at the PFP.[364]

18   Plaintiffs do not cite any other expert as supporting

19 Klementiev's ARF methodology. Without any apparent expert

20 support, **plaintiffs' counsel** offers what they believe is

21 evidentiary support for Klementiev's ARF values. This includes

22

---

23   [364] Even if Klementiev had specifically referred to
    Mishima's Table II during the course of his deposition and
24 offered a scientifically defensible explanation for his use of
    it, that may not have been enough. The purpose of exchanging
25 expert reports is to let opposing counsel know about what the
    expert intends to testify, and to prevent the expert from
26 changing his opinion as the litigation progresses. Klementiev
    made no mention of Mishima's Table II in any of his expert
27 reports.

28

ORDER RE SUMMARY JUDGMENT-   **459**

1 an attack upon Mishima's analysis and HEDR's plutonium analysis.

2 A fundamental problem, of course, is that nowhere in his reports

3 does **Klementiev** attack Mishima's methodology. Indeed, he

4 specifically relies upon the 7% figure as "given" to him by

5 Mishima. Counsel cannot fill voids in the expert record.

6 Besides that, Klementiev's analysis is not made any more

7 scientifically reliable merely by virtue of purported

8 deficiencies in Mishima's experiments or HEDR's analysis.

9   Plaintiff's counsel assert Mishima did not consider actual

10 operating conditions or operating documents in arriving at his

11 conclusions, but considered only his laboratory experiments.

12 Counsel cite passages from Mishima's deposition testimony wherein

13 he acknowledged the intent of his experiments was to determine

14 airborne release during an accident, specifically a fire.[365]

15 Mishima testified his team did not consider looking at process

16 parameters to determine what should be done in the experiments to

17 represent an accident. (Plaintiffs' Response Br. at p. 31).[366]

18
_____

[365] If Klementiev's goal was to simulate operating
19 conditions, one has to question why he relied on Mishima at all.
   The court assumes he relied on Mishima because it was the best
20 data available.

21   [366] At his deposition, Mishima testified he did not consider
   his Table II results to be "representative" of how plutonium
22 particles might become entrained during a fire. Therefore, he
   moved on to a different type of experiment where there was no
23 direct airflow upon the sample. The results of this experiment,
   of course, are reported at Table VI. (Mishima Depo. at pp. 190-
24 91, Foulds Ex. 224). Mishima explained it this way:

25   If you have a fire . . . the air does
              not impinge on the material; if it did,
26   you would put out the fire . . . you
              can blow out a fire using very rapidly
27   blowing air.
28
ORDER RE SUMMARY JUDGMENT-   460

1   Counsel quote from a whole series of operational documents

2   (Plaintiffs' Response Br. at pp. 34-37) which they claim were

3   never reviewed by Mishima, but were "taken into account by

4   Klementiev." However, counsel do not state where or how any of

5   these documents were taken into account by Klementiev in his

6   expert reports. Counsel do not cite to any deposition testimony

7   wherein Klementiev explains how he used such documents. Although

8   plaintiffs' counsel may contend Mishima's Table VI results do not

9   accurately reflect operating conditions, the fact is Klementiev

10 relied upon them (as it turns out, inappropriately) for his

11 "process analysis."

12   Plaintiffs contend Mishima's entrainment rate does not

13 square with empirical data showing the amount of plutonium powder

14 found in the PFP ductwork. Plaintiffs claim that for the fiscal

15 years 1959-1962, 16,381 grams of plutonium particle was flushed

16 from the 234-5 building 26-inch vacuum system. According to

17 plaintiffs, Mishima's entrainment rate of 87 ppm for all Z-plant

18 processes[367] generates a figure of only 2.63 kg for all the

19 production years from startup through 1962, whereas by the end of

20 _____

21   And so normally, when you are heating
            things, the vapors and the convection
            are the only forces that will lift

22   the particles that are ejected from the
            mass in any . . . short distance and get

23   carried into the flow.

24 (Id. at p. 188).

25   [367] In his affidavit, Mishima states "my research and the
    research of others would indicate that a scientifically-based

26 long term airborne release fraction for the PFP chemical
    processing operations should be in the area of 87 parts per

27 million." (Mishima Affidavit at p. 4, Paragraph 12).

28
    ORDER RE SUMMARY JUDGMENT-   461

1 1962, over 16 kg (16,381 grams) had already been flushed out of

2 the ducts. Plaintiffs do not cite any report from Klementiev (or

3 any other expert) ascribing significance to this purported

4 inconsistency, in particular that it somehow bolsters the

5 reliability of **Klementiev's** 20,000 curie release estimate, an

6 estimate derived in part from Mishima's work.

7   Plaintiffs assert operational documents show the average

8 dust entrainment rate for the fluorinator and calciner would have

9 exceeded 15 grams per hour. One fluorinator alone, say

10 plaintiffs, would generate 360 grams per day during a continuous

11 24 hour operation (15 x 24). "Assuming" a 300 day year to allow

12 for maintenance interruptions, 108 kg of plutonium dust would be

13 produced. In light of this, plaintiffs assert Mishima's

14 calculations are "patently ridiculous as applied to Hanford

15 reality." Plaintiffs note that applying Klementiev's 7% ARF to

16 an average 4,750 kg per year production generates a total of 332

17 kg entrainment per year.[368] According to plaintiffs:

18   Considering just the fluorinator alone is
           producing 108 kg/year powder entrainment,
19   and with the calciner producing an equal
           amount, and adding the **probably** comparable
20   amount of oxalate powder entrainment after
           it has dried and is being dumped from a
21   hopper into a calciner, for an approximate
           total of 324 kg/year, [Klementiev's]
22   estimate of 332 kg/year comes out remarkably
           close to the measured production averages
23   of 15 grams/hour.

24 (Plaintiffs' Response Br. at p. 38)(Emphasis added).

25 _____

26   [368] This is **before** any entrained particles hit the filters.
   Indeed, plaintiffs' counsel concede that "[o]f course most of
27 this dust or powder was caught in the filters."

28 **ORDER RE SUMMARY JUDGMENT-**   **462**

1   Once again, plaintiffs do not cite any expert support for

2   this analysis. There certainly is no indication Klementiev used

3   this as an example to justify his 20,000 curie estimate. Beyond

4   that, Klementiev's 7% ARF is not a scientifically reliable figure

5   since he incorrectly interpreted the source for that figure

6   (Mishima). If it were scientifically appropriate to use

7   Mishima's Table VI result of 0.82% (8200 ppm) entrained on the

8   filter and apply it to an average production per year of 4,750

9   kg, the total is approximately 39 kg. That is significantly less

10   than 332 kg.

11   Plaintiffs contend that in his expert report prepared for

12   defendants, Mishima "heedlessly" applied his experimental

13   airborne release fractions to the actual operating conditions in

14   the Z-Plant. They further assert that Mishima deliberately

15   omitted from his DOE Handbook[369] summarizing his experiments

16   "the most important and frequently encountered stress during

17   actual processing, namely, direct airflow over the surface or

18   impingement combined with vibration."

19   The subject of this motion in limine is Klementiev, not

20   Mishima. Mishima's report is not at issue. Likewise, Mishima's

21   DOE Handbook is irrelevant. The fact is Klementiev derived his

22   7% ARF (and errantly so) from an experiment in which airflow was

23   not directly aimed at the powder. Klementiev did not rely on the

24   results from Mishima's experiment involving airflow aimed

25   _____

[369]   **DOE Handbook: Airborne Release Fractions/Rates and**
26   **Respirable Fractions for Nonreactor Nuclear Facilities, Volume I:**
     Analysis of Experimental Data and Volume 2:   Appendices.   (DOE-
27   HDBK-3010-94)(December 1994)(Defendants' Ex. 166).

28   **ORDER RE SUMMARY JUDGMENT-   463**

1 directly at the powder. Nor does Klementiev ever say why it

2 would have been appropriate to do so.

3   Plaintiffs criticize Mishima for using "an

4 uncharacteristically large size range of plutonium oxide of 15-40

5 microns in his experiments instead of the submicron sizes

6 normally encountered in the oxide dusts." According to

7 plaintiffs, "[by] only testing the larger plutonium oxide

8 particles, Mishima promotes a result that only a small fraction

9 of the powder would climb vertically to reach the filters

10 situated at the top of his chimney." In other words, plaintiffs

11 say Mishima's ARF values are not accurate.

12   Once again, it is not Mishima's ARF values which are the

13 subject of this motion in limine. It is Klementiev's values. If

14 Mishima did not measure submicron particles in his powder

15 experiments, that apparently did not bother Klementiev because he

16 took his 7% ARF value directly from Mishima's powder experiments.

17   For all of the reasons set forth above, the court finds

18 Klementiev's 7% ARF value for 234-5Z processing was not derived

19 by scientifically reliable means.

20

21   -- 231-Z and 234-5Z Casting Operations

22   Casting involves melting plutonium metal and pouring it into

23 castings to produce the shapes necessary for machining. For both

24 231-Z Casting and 234-5Z Casting, Klementiev uses a 3% ARF.

25   In his February 1997 report, Klementiev quotes from a July

26 3, 1996 interview with former Hanford operator Raymond King:

27   [T]he metal would undergo at least one casting,

28 **ORDER RE SUMMARY JUDGMENT-   464**

> and three if alloy was being made. Each casting
> would require the plutonium to be melted into
> liquid form . . . . When so heated the liquid
> plutonium would bubble up like boiling oil,
> sputtering and releasing gas-like vapors. Perhaps
> as much as 5% would become temporarily airborne.

(Klementiev 1997 Report at p. 9).

Klementiev also quotes from a 1996 interview with "A.B." that "there would be some loss between the weight of the buttons and the casting weight [and] this difference might average about one percent of the original weight of the buttons." (Id. at p. 10). Relying on these two sources, Klementiev arrived at an ARF (aka "plutonium entrainment rate) of 3% for 231-Z Casting (within a range of 0 to 6%). (Id.).[370] Klementiev uses the same sources to arrive at the same ARF (3%) for 234-5Z Casting. (Id. at p. 16).

Defendants contend the quote from King provides no scientific support for Klementiev's 3% ARF. They note that Klementiev omitted to quote King in full. Although King stated that "[p]erhaps as much as 5% would become **temporarily** airborne," he followed by saying the material would then "condense on various colder surfaces," after which "[w]e would collect the condensate and get **credit** for it." (Report of Interview with Raymond King at p. 3, Defendants' Ex. 172)(Emphasis added).

According to defendants, the balance of King's statement makes clear he was not saying that 5% would remain airborne and enter the ventilation system. In an affidavit supplied by

---

[370] It appears the 3% figure splits the difference between the 5% reported by King and the 1% reported by "A.B."

ORDER RE SUMMARY JUDGMENT-   465

1   defendants (Defendants' Ex. 171), King confirms as much: "I did

2   not state or mean to imply that 5% of the plutonium would **remain**

3   airborne and be taken up into the ventilation system." (Id. at

4   p. 2)(Emphasis added). He adds that his 5% estimate "was not and

5   is not an airborne release fraction . . . ." (Id.)

6   At his deposition, Klementiev testified that he

7   "interpreted" the wording "temporarily airborne" as meaning

8   "entrained." However, he never confirmed this with King. When

9   confronted with King's statement that after becoming temporarily

10 airborne, the material condensed on colder surfaces, Klementiev

11 admitted he had not contacted King to determine the amount of

12 condensate. Klementiev did not deny it was appropriate to

13 consider the amount of condensate. However, Klementiev states he

14 "took some precautions when he read this 5 percent . . . and

15 reduced it down to 3 percent." (Klementiev Dep. at pp. 777-78).

16   Plaintiffs say that "[a]s to casting[,][Klementiev] took an

17 initial entrainment estimated by witnesses as 5% and applied a

18 reduced figure of 3% (since some of the entrainment would remain

19 in the casting furnace and would not reach the filters, and used

20 a range of 0% to 3% for his uncertainty analysis)." (Plaintiffs'

21 Response Br. at p. 15).[371] However, plaintiffs do not cite to

22 any of Klementiev's reports or to any of his deposition testimony

23 wherein he explains and justifies this rationale. Indeed, in the

24 deposition testimony cited above (pp. 777-78), Klementiev did not

25 elaborate why he took "precautions" and reduced the 5% to 3%.

26 _____

    [371] As noted above, Klementiev's uncertainty range was not
27 0% to 3%, but 0% to 6%.

28 **ORDER RE SUMMARY JUDGMENT-   466**

1   In any event, the court agrees with defendants that King's

2   statements are no more support for a 3% figure than they are for

3   a 5% figure. Klementiev does not know the amount of the

4   condensate collected by King and his staff. King certainly does

5   not in any way advocate a 3% ARF. In his affidavit, he states he

6   would defer to the expertise of Mishima in estimating ARFs for

7   these processes. (Defendants' Ex. 171 at p. 2). Without King,

8   Klementiev has no support whatsoever for his 3% ARF for the 231-Z

9   and 234-5Z casting processes.[372]

10

11   -- **231-Z Burning Operations**

12   Plutonium-bearing scrap was burned at 231-Z which, according

13   to Klementiev, caused formation of fine particulate plutonium

14   oxide. For this process, Klementiev used an ARF of 1.46% which

15   he derived from "burn" experiments conducted by Mishima in

16   1965.[373] (Klementiev October 1996 Report at p. 5; February

17   1997 Report at pp. 11-12).

18   Defendants contend Mishima does not support Klementiev's

19   ARF. In his 1965 report, Mishima concluded from his experiments

20   that the ARF for burning operations ranged from approximately 3

21    $(2.8 \times 10-6)$ to 50 $(5.2 \times 10-5)$ parts per million. (Mishima 1965

22   at p. 10; Mishima Affidavit, Defendants' Ex. 174 at p. 5).

23   _____

        [372] Klementiev's reference to an interview with "A.B." for

24   a 1% airborne loss cannot, by itself, support the 3% figure.
     King is essential for Klementiev's 3% ARF.

25
        [373] Mishima, J., "Plutonium Release Studies: I. Releases

26   from Ignited Metal," (BNWL-205) (December 1965) (Defendants' Ex.
     175).

27

28   **ORDER RE SUMMARY JUDGMENT-   467**

1 Klementiev acknowledged this (Klementiev Dep. at p. 710),

2 although his 1.46% ARF equates to 14,600 ppm, considerably higher

3 than 3 to 50 ppm.

4   Klementiev agreed that Mishima's experiments are properly

5 referred to as an "air measurement approach." (Klementiev Dep.

6 at pp. 703-04). Mishima set out to determine how much plutonium

7 would be released to ventilated air if plutonium metal was

8 burning. Experimental plutonium metal was placed in a quartz

9 container and burned while ventilation air was supplied.

10 Particles in the ventilated air were removed with a filter and

11 the filter was then analyzed to determine how much plutonium was

12 released to the air. The amount of plutonium measured in the air

13 compared to the amount of the experimental plutonium was the

14 basis for Mishima's ARF (3 to 50 ppm). (Id. at pp. 702-03).

15   Klementiev's 14,600 ppm ARF was derived from a finding

16 reported by Mishima at p. 12 of his 1965 report:

17   After cooling, the residual material in the
           ignition boat and tube was collected and
18   weighed. Weight per cent oxide recovered
           ranged from 97.37 to 99.97, based upon the
19   weight of plutonium dioxide possible.

20 (Klementiev October 1996 Rpt. at p. 5). Based on the "before and

21 after" weight approach, Klementiev concluded the entrainment rate

22 (ARF) during oxidation and cooling ranged from 0.03% (100% -

23 99.97%) to 2.63% (100% - 97.37). Klementiev concluded it was

24 "reasonable to suggest" the "entrainment rate was of the order of

25 1%-2% or even higher." (Id.). An average of the 0.03% and 2.63%

26 figures produces 1.46%.

27   Defendants note that Mishima specifically rejects this type

28 **ORDER RE SUMMARY JUDGMENT-   468**

of approach for the purpose of determining airborne release

fractions and that had he intended to measure ARF by loss of

weight, he would have conducted "a much different experiment."

(Mishima Affidavit at p. 6, Paragraphs 19-20). In his 1965

report, Mishima stated:

> The apparent low oxide recovery may be
> partially due to the incomplete conversion
> of metal to the dioxide. Schinzlein and
> Fisher have also reported less than theoretical
> weight gain from the ignition of metallic
> plutonium in the air.

(Mishima 1965 at p. 12). In his affidavit, Mishima adds that the

"loss of weight can also be attributed to material that was not

recovered from the equipment used in the experiment." (Mishima

Affidavit at p. 5, Paragraph 18).

Without citation to any of their own expert reports

(including those of Klementiev) or to any expert affidavit

(including one from Klementiev), plaintiffs assert the "weight

difference" approach is scientifically appropriate for measuring

ARF. Instead, they cite Mishima's deposition testimony and

contend Mishima himself recognized the validity of the "weight

difference" approach. Plaintiffs cite deposition testimony from

Mishima in which he stated he tended to agree that if a small

percentage of the sample was not oxidized ("incomplete

oxidation"), it would change the weight difference by only a very

small amount. (Mishima Dep. at p. 84).

Plaintiffs' failure to cite any expert support for their

argument is alone enough to warrant its rejection. Furthermore,

plaintiffs' reference to Mishima's deposition testimony is hardly

ORDER RE SUMMARY JUDGMENT-   469

compelling evidence that Mishima considered the "weight difference" approach to be a valid method for calculating ARF.[374]

Plaintiffs criticize Mishima for using "just the results from his capture methodology[375] and ignor[ing] his before and after results." Plaintiffs reject Mishima's rationale that "the weight difference would have been so small that [he] could not detect it on an analytical balance . . . ." (Mishima Dep. at p. 107). According to plaintiffs, this "presupposes a result" and is "a contradiction in terms" because:

> [I]f the analytical balance is too imprecise
> to precisely measure the residue left after
> the experiment, it is necessarily too imprecise
> to precisely measure the beginning weight of the
> sample, but it's against this beginning weight
> as compared to what he captures on the filters
> that Mishima calculates his percentage of weight
> loss by entrainment!

(Plaintiffs' Response Br. at p. 24).

Here again, the plaintiffs do not cite to any expert report or affidavit in support of an argument which clearly warrants such. When pressed on the issue at his deposition, Mishima explained that the analytical balance "is good to ten to the minus four of the initial weight of one gram, roughly," and "[i]f

---

[374] In his deposition testimony, it does not appear Mishima identified what he considered to be the percentage of the sample not oxidized. Defendants contend Mishima explained the range was somewhere between 2 and 5% which is more than enough to account for the 0.03% to 2.63% Klementiev claims was entrained. The fact Mishima may not have identified the amount he thought was not oxidized does not make the criticism from plaintiffs' counsel any more valid.

[375] Referring to what was captured on the filter pursuant to the "air measurement" approach.

ORDER RE SUMMARY JUDGMENT–   470

the amount of material airborne were ten to the minus five, it is

beyond the capacity of the scale to measure that." (Mishima Dep.

at pp. 136-37).

Plaintiffs say Mishima ignored the "before and after" weight

results of the spontaneous combustion of a plastic bag of

plutonium shavings which showed a loss of 4.6%. Plaintiffs

assert this is of the same magnitude as the 2.63% weight loss

which occurred in Mishima's burn experiments. In his October

1996 report, Klementiev referred to this and quoted from Bell,

R.S, "Plutonium Metal Turnings Fire," (HW-33125)(September 20,

1954)[376]:

> On July 27, 1954 at about 2:15 p.m., 965
> grams of Pu alloy . . . were being removed
> from the process line in room 235 by two
> process operators using the plastic bag
> technique . . . Soon after . . . smoke appeared
> inside the plastic bag and then fell to the
> floor where they continued to burn until they
> were completely oxidized . . . . To date 95.4%
> of the original metal content in turnings
> has been recovered.

(Klementiev October 1996 Report at p. 5).

Although Klementiev cited this in his report, he made clear

at his deposition that his ARF was based on the Mishima data

which he described as "firm data I can rely on, the before-and-

after measurement." (Klementiev Dep. at p. 722-23). Defendants

argue Bell 1954 provides no scientific basis for an assumed ARF

of 1.46% because it was an accident and not a controlled

experiment, and because there was no attempt to include the

amount of plutonium vacuumed up while cleaning the area.

---

[376] Bell 1954, Foulds Ex. 237.

ORDER RE SUMMARY JUDGMENT-   471

Whatever the case, it is not important since it is clear from
both Klementiev's report and his deposition that the 1.46% ARF is
derived from Mishima's work.

Plaintiffs say Klementiev utilized the "before and after"
results of Mishima's burn experiments because "he had no
confidence in the other results that were based upon measuring
the amounts captured on the apparatus filters."[377] Plaintiffs
assert Hanford documents are replete with indications these
filters are transparent to very small particles.

In his October 1996 and February 1997 reports, Klementiev
discusses filtering efficiency. It appears he arrived at a
conclusion that the filters used in the plutonium finishing plant
ventilation/filtration system were the same as, or similar to,
those employed in Mishima's experiments and therefore, suffered
from the same alleged defects. (Klementiev 1997 Report at pp. 10
and 12). At his deposition, Klementiev indicated he agreed the
"air measurement" approach is the standard method for performing
and interpreting ARF experiments, provided "all the particulate
sizes is covered or caught by the filter. . . ." (Klementiev
Dep. at p. 704). Klementiev stated it was his belief Mishima had
not covered the full spectrum of particle sizes. Klementiev
based this belief on his opinions regarding membrane filter
efficiency. (Id. at pp. 710-11).

Filter efficiency is discussed infra. However, the court
must say it is not readily apparent how purported filter

_____

[377] This refers to the Millipore filters used by Mishima in
his "burn" experiment.

ORDER RE SUMMARY JUDGMENT-   472

1 deficiency makes the "weight differential" approach any more

2 scientifically reliable. The simple fact remains that plaintiffs

3 have not cited any expert support in favor of that approach.

4 Furthermore, Mishima explicitly rejects that approach.

5   In other words, even if Mishima's ARF of 3 to 50 ppm is

6 somehow inaccurate because he used defective filters, how does

7 that specifically confirm Klementiev's ARF of 14,600 ppm based on

8 the "weight differential" approach, an approach which has nothing

9 to do with filters and air measurement, but only with what is

10 left in the quartz container? Plaintiffs and Klementiev may

11 contend there are defective filters and therefore, unaccounted

12 particles which as a general proposition should make the ARF

13 higher. However, that does not mean the "weight differential"

14 approach is scientifically reliable for calculating ARF. The

15 "weight differential" approach is not made scientifically valid

16 merely by virtue of purported inaccuracies or deficiencies in

17 Mishima's "air measurement" approach.

18   Klementiev's 1.46% ARF for the burning of plutonium-bearing

19 scrap is not based on sound scientific methodology.

20

21   **-- Summary**

22   Klementiev's ARFs are a critical component of his process

23 analysis. Without them, his entire analysis, and the 20,000

24 curie release estimate produced by it, is rendered worthless.

25 Klementiev's ARFs are not scientifically reliable. On this basis

26 alone, the court is justified in excluding the 20,000 curie

27 release estimate.

28 **ORDER RE SUMMARY JUDGMENT-   473**

(iii)   Filter Efficiencies (Component of Process Analysis)

In arriving at his 20,000 Ci release estimate, Klementiev calculated that 3.25% of the plutonium particles which made their way into the ventilation system at PFP would penetrate the HEPA (High Efficiency Particulate Air) filters. There were two sets of these filters. Klementiev's filter penetration value of 3.25% translates into a filter efficiency of 82%. The first set of filters removes all but 18% of the plutonium which reaches it (100-18=82) and the second set of filters removes all but 18% of the plutonium that gets through the first set (18% of 18%=3.24%). (Klementiev Dep. at pp. 798-801).[378] Klementiev justified his reduced filter efficiency because of the effect of "small particles." (Klementiev October 1996 Report at p. 16).

Klementiev begins his filter efficiency analysis with a document that provides measurements of plutonium within the PFP ventilation system. L.A. Mahoney, et al., "Literature Review Supporting Assessment of Potential Radionuclides in the 291-Z Exhaust Ventilation," (August 1994). Based on this document, Klementiev arrived at a filter efficiency estimate of 98.2%. Klementiev stated "[t]he first approximation of HEPA filter efficiency equal to 98.2% seems realistic . . . when the particles composition in the airflow is suggested to be homogenous." (Klementiev October 1996 Report at p. 12). According to defendants, had Klementiev used a 98.2% filter

---

[378] In the uncertainty analysis contained in his February 1997 report, Klementiev calculated a distribution function for the release factor from HEPA filters of 0% to 35%. (Klementiev 1997 Report at p. 13). 18% is an approximate median value.

ORDER RE SUMMARY JUDGMENT-   474

1 efficiency, it would have reduced his release estimate a hundred

2 fold, from 20,000 Ci to 200 Ci. However, based on his belief

3 that the particle composition was not "homogenous" (that there

4 were smaller particles), Klementiev reduced this filter

5 efficiency value.

6   Relying on several different Hanford documents, as well as

7 Jervis' 1996 report, Klementiev found:

8   Under more realistic conditions, when the
            proportion of small particles entering the
9   first filter were assumed to be equal to 50%,
            and for those particles the filter efficiency
10   were suggested to be 60%, then the total amount
            of plutonium released to the atmosphere from
11   231-Z and 234-5Z plants would be 23.8 kg/yr.

12 (Klementiev October 1996 Report at p. 18).[379]

13   There appears to be some uncertainty on Klementiev's part as

14 to what exactly he means by "small" particles. In his October

15 1996 report, he quoted from Mahoney that the "deposition velocity

16 of particles below 10 [microns] AED [aerodynamic equivalent

17 diameter] is so low . . . that only a fraction of these particles

18 are likely to be deposited in a several-minute travel time."

19 (Id. at p. 13). In a footnote, Klementiev defined "small"

20 particles as less than 10 u AED." (Id., note 14). When asked at

21 his deposition if it was correct that he was assuming "small"

22 particles to be those less than 10 microns AED, he stated he was

23 "not sure" and that it was probably supposed to be 1 micron.

24 Klementiev indicated that when he had discussed the matter with

25 Jervis "probably a couple of years ago," it "was 1 micron, not 10

26 _____

27   [379] See also Klementiev February 1997 Report at p. 10.

28 **ORDER RE SUMMARY JUDGMENT-   475**

1   microns."   (Klementiev Dep. at pp. 805-06).

2   Frankly, this does not reflect favorably on Klementiev's

3   knowledge about "small particle" theory.   Jervis' 1996 report

4   refers to "submicron" particles, as does plaintiffs' response

5   brief.   Therefore, "less than 1 [micron]" makes more sense if one

6   is referring to "submicron" particles.

7   Defendants contend Klementiev's filter efficiency theory is

8   based on an unsubstantiated assumption that the smaller the

9   particle, the less efficiently it will be collected on the HEPA

10   filter.   They also note that Klementiev could not cite any

11   scientific publication supporting his assumption about HEPA

12   filtration efficiency.   (Klementiev Dep. at pp. 803-05).

13   Defendants cite a report prepared by one of their experts,

14   Dr. Melvin W. First, "Critique of Reports Estimating Losses of

15   Plutonium from Hanford Operations."   First, a Professor of

16   Environmental Health Engineering at Harvard School of Public

17   Health, discusses the collection of particles on fibrous filters.

18   One way in which particles are caught is filtration by

19   interception.   This is where the particles hit the filter fibers

20   in their "inertial path."   First states that particles larger

21   than 1 micron AED "exhibit inertia so that when the conveying air

22   stream must bend to pass over and under a fiber these particles

23   tend to continue on a **straight path** and make contact with the

24   fiber and remain."   First explains that the larger the AED of a

25   particle, the more likely it will be collected by the fiber, but

26   "[f]or particles substantially below 1 [micron] AED, their

27   inertia is small and filtration **by this means** [filtration by

28

ORDER RE SUMMARY JUDGMENT-   476

interception] is inefficient." (First 1997 Report at p. 4,

Paragraph 12)(Emphasis added).

However, according to First, **filtration by diffusion**

(Brownian diffusion) is more effective for smaller particles.

First says:

> . . . these small particles exhibit a random
> motion, called Brownian movement, as a result
> of continuous bombardment by the energenic
> air molecules in which they are suspended that
> causes them to dither in three dimensions around
> a streamline on which they are being conveyed
> by the air. When the air streamline passes near
> a filter fiber, Brownian motion causes them to
> strike the fiber, where they are retained. As
> small particles diminish in AED, their Brownian
> motion becomes more vigorous and their likelihood
> of striking a nearby filter becomes greater. **In
> other words, as small particles substantially
> less than 1 [micron], get smaller, they become
> easier to collect (more efficient) in fibrous
> filters.**

(Id.)(Emphasis added).

Plaintiffs do not respond to defendants' argument that

Klementiev failed to consider these concepts of filtration theory

as enunciated by Dr. First. According to plaintiffs, defendants

agreed to withdraw their references to Dr. First's expert report.

(Plaintiffs' Response Br. at p. 2, n. 1). Indeed, the paragraph

of Dr. First's report to which defendants refer- Paragraph 12

explaining filtration by interception and by diffusion- was

actually stricken by this court. In its October 1997 "Order

Granting Reconsideration, In Part" (Ct. Rec. 1065), the court

stated that paragraphs 10-16 of First's report were intended as a

critique of Jervis' 1997 report. Because Jervis' 1997 report had

been previously stricken, the court found there was no reason for

ORDER RE SUMMARY JUDGMENT-   477

First to rebut Jervis' 1997 report.   (Order at pp. 11-12).

In their reply brief, defendants seemingly attempt to get around using First's expert report by citing to work he performed at an earlier date which discussed Brownian motion of small particles and how filtration by diffusion is more effective for picking up such particles. First, "Removal of Airborne Particles from Radioactive Aerosols," in Goossens, et al., **Treatment of Gaseous Effluents at Nuclear Facilities** (1991) (Defendants' Ex. 209 at p. 27).[380]

The court is not impressed with plaintiffs' failure to make any attempt to substantively address the filtration concepts explained by Dr. First. Although paragraph 12 of First's expert report has been stricken, the court notes that Klementiev, in his February 1997 report, used First as a reference. The court believes the points made by First with regard to filtration theory are standard concepts which should be considered in assessing the reliability of **Klementiev's** work, as opposed to **Jervis'** work.

In his February 1997 report, Klementiev quoted as follows from Dr. First's July 1990 "Draft Report on Filter Systems at Rocky Flats Plant"[381]:

> J.A. Hayden and R.W. Woodward performed three
> plutonium size analyses at RFP in 1977 . . .
> using the aerosol from some point in a glove
> box exhaust system. They used four stages
> of HEPA filter paper mounted in series . . . .
> The numbers cited in the report show efficiencies

---

[380]   See Defendants' Reply Brief at p. 33.

[381]   Hereinafter, "First 1990."

ORDER RE SUMMARY JUDGMENT-   478

1   of 78% for stage 1; 48% for stage 2, and 49% for
     stage 3.

2

3   (Klementiev 1997 Report at p. 10).   Klementiev asserted this

observation by Hayden and Woodward was "consistent" with his

4

estimate of a 74.1% filtering efficiency, specifically where it

5

was assumed: 1) that half of the activity entering the filtering

6

system could be considered as associated with "regular" size

7

particles (98.2% of this activity would be trapped by the

8

filter); and 2) the other half of the activity entering the

9

filtering system are "small" size particles with a release factor

10

of 50%.   (Id.)[382]

11

     Defendants contend Hayden and Woodward provide no support

12

for Klementiev's estimates of filtering efficiency.   They assert

13

Klementiev omitted portions of the quote from First 1990 to make

14

it look as though Hayden and Woodward were discussing the

15

efficiencies of HEPA filters.   The full quote from First is as

16

follows:

17

18   Although it would be highly desirable to verify
            that the existing sampling line diameters, lengths,
            and sampling rates give minimum sampling losses (or

19   to be able to modify the system to achieve this
            desirable status), there is an essential piece of

20   information for such an analysis that is currently
            missing, namely, **the particle size distribution of**

21   **the aerosols being sampled.**

22   J.A. Hayden and R.W. Woodword [sic] performed three
            plutonium size analyses at RFP [Rocky Flats Plant]

23   in 1977 (Plutonium Particulate Penetration and Size
            Studies, RFP-2635) using the aerosol from some point

24   in a glove box exhaust system.   They used four stages
            of HEPA filter paper mounted in series **in separate 37**

25   **mm commercial filter cassettes.**   Two samples gave a

26   ───────────────────────

      [382]   See Column G at Row 11 of Table 3 of Klementiev's

27   October 1996 Report, p. 15.   The result is 0.741.

28

ORDER RE SUMMARY JUDGMENT-   479

1   count mean diameter CMD of 0.19 u for the filter stage
     and the third gave a CMD of 0.11 um. The numbers cited
2   in the report show efficiencies of 78% for stage 1;
     48% for stage 2; and 49% for stage 3. The
3   investigators were obviously dealing with a severely
     leaking cassette system of filter holders and the data
4   on stages 2, 3 and 4 are useless. **However, the numbers
     cited for particle sizes on stage 1 may be close to
5   the mark.**

6   (First 1990 at p. 6, Defendants' Ex. 167)(Emphasis added).

7   According to defendants, it is clear that Hayden and

8   Woodward were studying 37 millimeter commercial HEPA filter

9   cassettes and not the large HEPA filters used at PFP. At his

10   deposition, Klementiev was asked whether his quote from First

11   related to HEPA filters with the same dimensions and

12   characteristics as those used at PFP. Klementiev responded: "I

13   should think so." Klementiev acknowledged he had omitted the

14   language about "37 mm commercial filter cassettes." Asked how

15   large the filters were at PFP, Klementiev stated he had never

16   seen them, but from looking at photographs his impression was

17   they were about "half a meter by half a meter . . . . [p]robably

18   slightly more." (Klementiev Dep. at pp. 809-11). Half a meter

19   is equivalent to approximately 20 inches (1 meter = 39.37 in.),

20   substantially more than 37 millimeters which is equivalent to

21   1.48 inches (37 mm x 0.04 in.).

22   Defendants say Klementiev omitted the portions of the quote

23   which show First was examining the data in regard to particle

24   sizes, not the efficiency of 37 mm commercial filter cassettes.

25   Indeed, certain language indicates First was focusing on particle

26   size- i.e. "number cited for particle sizes on stage 1 may be

27   close to the mark." According to defendants, First cited Hayden

28   **ORDER RE SUMMARY JUDGMENT-   480**

1 and Woodward only for their finding regarding the size of the

2 particles that hit the first filter (Stage 1) and rejected the

3 rest because of a "severely leaking cassette system" which

4 rendered useless the data on stages 2, 3 and 4.

5   In their response brief, plaintiffs turn a deaf ear to

6 defendants' argument. Their lack of response can only be

7 construed as a concession to the validity of the argument. At

8 his deposition, Klementiev acknowledged he not even read the

9 document, (Geer, et al., "Filter Testing and Development for

10 Prolonged Transuranic Service and Waste Reduction," (February

11 1977)), from which First pulled the Hayden and Woodward data.

12 Klementiev simply took from First 1990 what he thought supported

13 his position.

14   In his February 1997 report, Klementiev asserted that "[i]n

15 addition to the fact that the filtering efficiency of the real

16 HEPA filters was uncertain, there were difficulties in obtaining

17 undamaged HEPA filters." He added "[t]here were also

18 installation problems that **could** dramatically reduce HEPA

19 filters['] efficiency." (Klementiev 1997 Report at pp. 2-

20 3)(Emphasis added).

21   Klementiev cited a 1959 document, Thaxter, M.D., July 7-9,

22 1959, "Condition of Commercial High-Efficiency Filters upon

23 Receipt or Installation," Sixth AEC Air Cleaning Conference, U.S.

24 AEC Office of Technical Information. Klementiev quoted from

25 Thaxter regarding rejection of filter shipments because of media

26 rips and also "that inattention to details and inspection of

27 filterbank hardware, in assembly bolt pressures . . . can result

28 **ORDER RE SUMMARY JUDGMENT-   481**

1 in actual leakage around the filter and consequent pollution

2 downstream." Klementiev said this allowed for an assumption that

3 "the real filtering efficiency of the undamaged HEPA filters

4 installed in the system **might** be significantly lower than

5 99.97%."[383] (Klementiev 1997 Report at pp. 2-3)(Emphasis

6 added).

7   Defendants contend this information is of no significance to

8 Klementiev's filter efficiency values which are based on the

9 "small particle" theory, not filter testing. At his deposition,

10 Klementiev was asked whether the filters were tested before they

11 were installed in the PFP ventilation system. He answered that

12 as far as he knew, the filters were "visually inspected," but was

13 not aware "[w]hether they were tested and how they were tested."

14 (Klementiev Dep. at p. 813). Klementiev stated he had learned

15 from additional "declassified documents" that HEPA filters were

16 installed following visual inspection, even though it was known

17 the filters were damaged. However, Klementiev made clear "this

18 knowledge . . . **didn't make any changes in my report**." (Id. at

19 pp. 813-14)(Emphasis added). He added that with regard to the

20 filters, he preferred to "stay amateurish . . . in the sense that

21 I can't understand the physics of what is happening there."

22 Klementiev indicated he was deferring to Jervis on matters

23 related to filter efficiency. (Id. at pp. 814-15).

24   Plaintiffs say that "[w]hile Klementiev adjusts his average

25 filter efficiency for small particle sizes, he observes that

26 _____

     [383] The source of this 99.97% figure is Blasewitz, discussed
27 infra.

28 **ORDER RE SUMMARY JUDGMENT-   482**

filter efficiencies would also be compromised because there were 'difficulties in obtaining undamaged HEPA filters' and additional problems with installation." (Response Br. at p. 45). Plaintiffs cite a number of sources pertaining to shipment of defective HEPA filters (including Thaxter), installation of defective filters, and improper installation of filters. (Id. at pp. 46-47).

Nonetheless, as noted above, Klementiev makes clear this type of information "didn't make any changes in his reports." Klementiev's 82% filter efficiency is derived solely from his "small particle" theory, with his reference to Thaxter merely serving as an invitation to speculate about the actual numerical efficiency of the filters.[384]

In his October 1997 report, Klementiev's starting point was Mahoney's 98.2% filter efficiency estimate based on actual measurements of plutonium in the PFP ventilation system. The **only** basis Klementiev offered for reducing that figure was the "small particle" theory. At p. 16 of his October 1996 report, Klementiev asserted the 98.2% figure neglected the effect of reduction filtering efficiency pertaining to "small particles" and were that taken into account, "it is likely . . . the HEPA filter efficiency would be as low as 92% or lower." (See also p.

---

[384] In his October 1996 report at p. 6, Klementiev stated that historical records suggested the efficiency of the 231-Z filtering system was lower than suggested by the manufacturer due to leakage of gaskets, malfunctioning of the filters, and "probably" due to not accounting for the submicron size range of the particles. However, Klementiev acknowledged that **"numerical estimation of the actual filtering efficiency of the plutonium-bearing materials in 231-Z is still not done."** (Emphasis added).

ORDER RE SUMMARY JUDGMENT-   483

10 of February 1997 Report discussing "uncertainty" of filter

efficiency).

In his very first report- March 1996 - Klementiev accepted a

99.76% rate of efficiency for the fiberglass filters used at the

separations plants (not the Z-Plant), instead of HEDR's 99%

estimate.   (Klementiev Dep. at p. 589).   For the 99.76% figure,

Klementiev used Blasewitz, "Comments Re: Radionuclide Releases

to the Atmosphere from Hanford Operations," (December 1993).

(Klementiev March 1996 Report at p. 5).   Defendants point out

that during his deposition, Klementiev said he would not reject

the idea that the HEPA filters at the Z-Plant were more efficient

than the fiberglass filters at the separations plants.

(Klementiev Dep. at pp. 305-06).   Thus, say defendants,

Klementiev admitted the HEPA filters had to be better than 99.76%

efficient, contradicting the 82% efficiency assumed for his

20,000 Ci estimate.

Defendants' argument has merit.   As early as his March 1996

report, Klementiev considered what Jervis had said about

submicron particles in his [Jervis'] 1996 report.   Klementiev

quoted Jervis' 1996 report[385] to the effect that "[p]lutonium

releases to the environment from the Hanford separations plants

and final product handling stacks were appreciably underestimated

(probably by at least an order of magnitude) and that plutonium

on tiny particles both eluded and penetrated stack filtration

devices and escaped detection."   Based on a March 1996 telephone

---

[385]   Jervis, "Reliability Assessment of Pu Release Estimates
at Hanford ('48-'80)," (March 1996).

ORDER RE SUMMARY JUDGMENT-   484

1 | conversation with Jervis, Klementiev stated that the particles

2 | were submicron in size and made up at least 50% of the activity

3 | not caught on air-sampling filters and therefore, not reported as

4 | stack losses. Klementiev concluded the total activity released

5 | to the atmosphere "was at least two times higher than it was

6 | measured and reported in the available historical records."

7 | (Klementiev March 1996 Report at p. 9).

8 | In his March 1996 report, Klementiev put forth three

9 | scenarios for estimating Z-Plant stack releases. Scenario C used

10 | a generic release ratio based on HEDR's cumulative plutonium

11 | release estimate of 1.78 Ci and considered a filtering efficiency

12 | of 99.76% (based on Blasewitz) to arrive at a total Z-Plant stack

13 | release of 0.15 Ci. (Klementiev March 1996 Report at p. 11 and

14 | Table 13 at p. 19). In Scenario D, Klementiev considered the

15 | same factors, plus **accounted for submicron particles** based on the

16 | information from Jervis' 1996 report. Therefore, Klementiev

17 | doubled his Z-Plant stack release to 0.30 Ci which was "two times

18 | higher" than 0.15 Ci. (Id. at p. 11 and Table 14 at p. 19).

19 | Finally, in Scenario F, Klementiev considered all of the factors

20 | in Scenario D, including the **accounting for missed submicron**

21 | **particles**. However, instead of using HEDR's cumulative release

22 | estimate, Klementiev used the information about MUF to calculate

23 | his "generic release ratio." This had the effect of upping

24 | Klementiev's Z-Plant stack release to 1,168 Ci (Klementiev's

25 | "second" release estimate). (Id. at pp. 11, 16, and Table 16 at

26 | p. 20).

27 | What is significant and problematic, according to

28 |

ORDER RE SUMMARY JUDGMENT-   485

1   defendants, is that Klementiev's filter efficiency estimate for

2   both the results of his March 1996 report (highest estimate 1,168

3   Ci) and his October 1996 and February 1997 reports (highest

4   estimate 20,000 Ci) was derived from the **same source:** Jervis'

5   1996 report. While it is true that in his latter reports

6   Klementiev started out with a new and lower baseline figure

7   regarding filter efficiency- 98.2% versus 99.76%- that alone

8   would hardly be enough to compensate for the substantial

9   difference in the estimates. Therefore, the difference could

10   only be attributed to a new interpretation of the "small

11   particle" theory set forth in Jervis' 1996 report.[386] Such a

12   new interpretation is not readily apparent from Klementiev's

13   October 1996 or February 1997 reports.

14   Unless there is some new and defensible interpretation of

15   Jervis' 1996 report, it indeed seems that Jervis' 82% efficiency

16   for HEPA filters at the PFP cannot be reconciled with

17   Klementiev's earlier 99.76% efficiency for fiberglass filters at

18   the separations plants. Both efficiencies are based on what

19   appears to be the same "small particle" theory and Klementiev

20   acknowledged he thought the HEPA filters were more efficient than

21   the fiberglass filters.

22   Plaintiffs do not, at least directly, confront this apparent

23   _____

[386]   Jervis prepared a March 1997 supplemental report which
24   was stricken by the court. Klementiev could not have considered
     this report since it did not yet exist when Klementiev prepared
25   his February 1997 report. At his deposition, Klementiev
     acknowledged he had not seen nor requested Jervis' March 1997
26   report. (Klementiev Dep. at p. 411). Klementiev's February 1997
     report cited only Jervis' March 1996 report. (Klementiev 1997
27   Report at pp. 18-19).

28   **ORDER RE SUMMARY JUDGMENT-   486**

1 contradiction. They do, however, suggest Jervis implicitly, if

2 not explicitly, approved of Klementiev's release estimates. This

3 has already been discussed. There is simply nothing to indicate

4 that Jervis specifically endorsed Klementiev's 82% efficiency for

5 the HEPA filters at PFP. At his deposition, Klementiev testified

6 he had not discussed this value (82%)[387] with Jervis.

7 (Klementiev Dep. at pp. 800-01).

8   In his 1996 report, Jervis discussed the stack filtration

9 systems at the separations plants (200 Area). He stated:

10   [S]pecific findings and other information
                recorded in . . . reports indicates that small
11   particles were discharged from Hanford stack
                after either by-passing or penetrating the
12   filtration systems. Studies of filter efficiencies
                for CWS-6 and fibre glass filters from the same
13   suppliers were done during the same period of
                time at Savannah and they reported efficiencies
14   as low as 90% for [plutonium and some fission
                products] and much lower for iodine (32%).
15
   (Jervis 1996 Report at p. 9).
16
          Jervis went on to specifically discuss the Z-Plant stacks:
17
                Because similar filter banks were used in
18   the Z-Plant stacks to minimize Pu losses as
                were used in the other 200 area stacks, some
19   emissions of small particle Pu from the Z-Plant
                were not unexpected although stack monitoring
20   claimed comparable efficiency.

21 (Id. at p. 12).

22   Jervis suggested such emissions were verified by Brabb's

23 1961 MUF analysis that "stack losses getting through the filters

24 probably account for 1 to 2 kilograms per year (of MUF)." (Id.).

25 One of Jervis' conclusions was:

26 _____

        [387] Derived from Figure 2 on Page 13 of Klementiev's
27 February 1997 Report.

28 **ORDER RE SUMMARY JUDGMENT-   487**

> PuO2 in finely divided form was undoubtedly
> by-passing and penetrating the stack ventilation
> systems in appreciable quantities and led
> investigators to the conclusion that (in 1961)
> as much as 1 to 2 kilograms per year were probably
> lost to the environment and contributed to the
> estimated 40-50 k of unaccounted losses that (sic)
> years.

(Id. at p. 14).

In his stricken 1997 report, Jervis discussed HEPA filter efficiencies and integrities (March 1997 Report at pp. 6-10). In his concluding paragraph, Jervis wrote that "[t]he public health and environmental consequences of releasing about **50** kg of submicron, dispersible and highly respirable plutonium at Hanford are considerable." (Id. at p. 15). This court struck Jervis' 1997 report because it did not materially alter the analysis contained in his March 1996 report which contended that decreased filter efficiencies verified the **40 to 50 kg** loss suggested by Brabb's analysis. The court found the "new" documents cited by Jervis in his 1997 report (including those pertaining to filter integrity) perhaps bolstered Jervis' prior analysis, but did not materially alter it. (Order Striking Supplemental Expert Report of Robert Jervis, July 14, 1997, Ct. Rec. 987).

Jervis may provide support for Klementiev insofar as concerns a most general proposition that HEPA filter efficiencies were lower than reported. However, the specific 82% filter efficiency value belongs solely to Klementiev. It is this value which produces the 20,000 Ci or 320 kg release estimate. At the very most, Jervis would endorse a significantly lower release estimate of 50 kg. Indeed, Jervis is not even very firm as to

ORDER RE SUMMARY JUDGMENT-   488

1 whether he believes 50 kg was released. Rather, his opinion is

2 that Brabb's MUF analysis, from which the 50 kg figure is

3 derived, is not beyond the pale considering HEPA filter

4 integrities and the inability of those filters to stop "small"

5 particles.

6   Plaintiffs contend Klementiev's "more realistic estimate" of

7 20,000 Ci is supported by various Hanford historical documents

8 regarding "small particles" and their ability to penetrate

9 filters. Plaintiffs assert the transparency of absolute filters

10 for .3 micron particles is part of the scientific literature,

11 citing L.A. Haack, "Analysis Of A Possible Influence Of Particle

12 Size On the Iodine Removal Efficiency of a Charcoal Filter,"

13 (October 1963).[388] As defendants point out, even the title

14 makes obvious that this analysis pertains to **iodine** on a **charcoal**

15 filter, not plutonium particles on a HEPA filter. Furthermore,

16 the report ("the paper") makes clear that "[t]he complete

17 penetration of the 0.3 micron particles, and below, and

18 efficiencies were **assumed** in this paper." Id. at p. 176

19 (emphasis added).

20   Plaintiffs cite additional documents which they say prove

21 Hanford contractors knew and accepted that submicron particles

22 pass through absolute filters. These documents include Borasky,

23 R., "Electronoscopic Particle Studies: I. Particles from an

24 Aerosol Containing Plutonium Oxide Dust," (HW-58673)(March 19,

25 1959); and Swain and Haberman, "Plutonium Emission Rates from

26 _____

27   [388] Foulds Ex. 186.

28 **ORDER RE SUMMARY JUDGMENT-   489**

1   Various Incidents in the 234-5 Building," (HW-89064)(May 10,

2   1961).   Swain and Haberman cite Borasky as indicating the size

3   range of particles hitting the filters was .05-7.0, whereas the

4   **filtered** particles ranged in size from .1 to .8.   Swain and

5   Haberman (1961) at Table 2 on p. 6.   The **filtered** range is taken

6   from Postma, et al., "Radioactive Particles in the 234-5 Building

7   Ventilation Exhaust," (July 13, 1959).

8   The point plaintiffs apparently try to make is that this

9   shows certain of the Borasky particles, those less than .1 in

10 size, were **not filtered** (i.e. they penetrated the filters).

11 Plaintiffs cite a passage from Swain and Haberman at p. 4

12 reporting that the aerosol[389] used for Curve A (Postma) "is

13 known to have passed through an absolute filter and the range of

14 particle sizes is very small."

15   Defendants contend Borasky is of no use because it does not

16 indicate that it relates to PFP or to air samples taken **upstream**

17 from the PFP filters.   Indeed, all Borasky says is that "[t]wo

18 Millipore filters exposed to an aerosol containing plutonium

19 oxide dust were submitted for electronoscopic analysis."   Borasky

20 1959 at p. 1.   Swain and Haberman confirm as much by reporting

21 that the aerosol in Curve B (Borasky) is of "unknown age and

22 history."   (Swain and Haberman at p. 4).   On this basis,

23 defendants persuasively contend plaintiffs have no evidentiary

24 basis for asserting the source of the Borasky particles is

25

26

_____

27   [389]   A suspension of fine solid or liquid particles in gas.

28 **ORDER RE SUMMARY JUDGMENT-   490**

1 upstream from the PFP filters.[390]

2   The plaintiffs contend there is legitimate scientific

3 dispute about the efficiency of the HEPA filters. That may well

4 be true. However, the question here is not whether such a

5 dispute exists, but whether Klementiev's analysis is

6 scientifically reliable.

7   Klementiev's 82% filter efficiency value is not

8 scientifically reliable. The lack of reliability is confirmed by

9 plaintiffs' failure to respond to a number of defendants'

10 arguments and the fact Jervis, whom plaintiffs claim as their

11 filter expert, did not endorse Klementiev's figures. Rejection

12 of Klementiev's analysis does not mean the filtering efficiency

13 dispute is put to rest for eternity. It only means that

14 Klementiev's work is not scientifically reliable.

15

16   **(iv) MUF (Material Unaccounted For)**

17   Plaintiffs suggest the fact 600 kilograms of plutonium was

18 "material unaccounted for" between 1956 to 1966 generally

19 confirms Klementiev's release estimates. Anderson 1977 at p. 2

20 reported that "a 600 kilogram plutonium inventory difference

21 associated with operations in the 234-5 Building, developed

22 during the period 1956 to 1966 while the facility was operated by

23 the General Electric Company and Isochem, Incorporated." In

24 _____

25 [390] Postma by itself is insufficient to make plaintiffs' argument that submicron particles were found upstream of PFP filters. Plaintiffs do not present the argument as such.

26 Rather, plaintiffs' argument is phrased in a way that the alleged significance of Postma is only revealed by comparing it to

27 Borasky, as was done in Swain and Haberman.

28 **ORDER RE SUMMARY JUDGMENT-   491**

other words, there was disagreement between the amount of

inventory as shown in **accounting** records and the amount of

material accounted for in the physical inventory. This is what

is known as a "Book-Physical Inventory Difference" or "B-PID."

Brabb 1961 at p. 1, attached to Anderson 1977.

Defendants argue the MUF data actually undermines

Klementiev's 20,000 Ci estimate. Klementiev testified it

"looked" like a "good estimate" that 70,000 to 80,000 total

kilograms of plutonium was processed at PFP. (Klementiev Dep. at

p. 826). Klementiev opined that 323 kilograms of this total

amount was released to the atmosphere. According to defendants,

Klementiev therefore necessarily assumes that more than half of

the 600 kg of MUF was released to the atmosphere (323 of 600 =

53.8%).

Defendants note once again that Anderson did not attribute

any of the MUF to stack releases. Brabb offered a "quantitative

speculation" that stack losses accounted for 1 to 2 kg per year.

(Brabb Rpt. at p. 5). For fiscal year 1961, the MUF or B-PID was

approximately 76 kg (75.8 kg). (<u>Id</u>. at p. 3). Defendants point

out that 1 to 2 kg amounts to 1.3 to 2.6% of 76 kg, which they

compare to the much larger 53.8% derived from comparing

Klementiev's total release estimate (323 kg) to the 600 kg of

MUF.

It must be pointed out that Klementiev's 323 kg total

release spans a greater period of time than just the 1956 to 1966

period for which the 600 kg MUF was reported. Therefore,

Klementiev might argue it is unfair to compare his 20,000 Ci

ORDER RE SUMMARY JUDGMENT-   **492**

release estimate to the MUF for just a ten year period (1956-66).[391] Although plaintiffs place much emphasis on the MUF issue, Klementiev did not base his 20,000 Ci release estimate upon it. The driving force for Klementiev's "process analysis" was the "small particle" theory and the ARF and filtration components thereof. Therefore, one cannot say Anderson and Brabb, by themselves, make Klementiev's 20,000 Ci estimate scientifically unreliable.

On the other hand, the court also fails to see how Anderson and Brabb provide any support for Klementiev's 20,000 Ci estimate. If one extends Brabb's "quantitative speculation" of 1 to 2 kg stack loss per year over the entire 10 years (1956-66), the result is 10 to 20 kg. 10 kg is 1.6% of 600 kg. 20 kg is 3.3% of 600 kg. Assuming Brabb's figures are correct and he is referring to total stack losses from all Z-plant operations, 1 to 2 kg over the approximately 20 to 25 years of PFP operation covered by Klementiev's analysis amounts to a maximum of 40 (20 x 2 kg) to 50 kg (25 x 2 kg) of stack releases. Note these are the figures at which Jervis arrived based on Brabb. Obviously, that is quite a bit less than 323 kg total release over the same period of time (20 to 25 years).

The defendants assert Klementiev's 20,000 Ci estimate is contrary to deposition testimony from plaintiffs' expert, Dr. Robert Goble, who opined that only 1 to 2% of MUF or missing plutonium was released to the atmosphere. (Goble Dep. at pp.

_____

[391] However, see footnote 396 _infra_.

ORDER RE SUMMARY JUDGMENT-   493

369-70).   1 to 2% is fairly close to the result from extending

Brabb's estimated 1 to 2 kg stack loss over a ten year period (10

kg is 1.6% of 600 kg; 20 kg is 3.3% of 600 kg).   Indeed, Goble

agreed that over "about an 11 year period," 1 to 2 kg was

released per year for a maximum loss of 22 kg from the PFP.   (Id.

at p. 369).[392]

According to defendants, Klementiev's 323 kg total release

estimate requires that 10,000 total kg became airborne and that

9,700 kg was deposited on the PFP filters and elsewhere in the

ventilation system.   At his deposition, Klementiev testified that

based on a "hypothetical" where 323 kg was released to the

atmosphere and the filter penetration value was 3.25%,   it

"looked" as though 10,000 kg would have arrived at the filters

(10,000 kg of the entire 70,000 to 80,000 kg processed over the

years).   Klementiev stated it "seem[ed] to be correct" that

approximately 9,700 would therefore be left on the filter (10,000

kg - 323 kg).   (Klementiev Dep. at pp. 824-25).[393]

---

[392]   Goble commented that "missing inventory is not generally
the best source of information" because, for example, he believed
there was a lot of plutonium stored in waste sites that had not
been released into the atmosphere.   (Goble Dep. at p. 368).
    In his 1961 memorandum, Brabb noted that "B-PID can arise
from process losses, from measurement uncertainties, from
accounting procedures or errors in accounting, and from
diversions (including thefts)."   (Brabb 1961 at p. 1).   Brabb
concluded that "B-PID's at Hanford represented **in part** a physical
loss of product" and that specifically, "[p]lutonium B-PID at Z
Plant arises in part from unrecorded but known diversions in the
burial of failed equipment."   (Id. at p. 4)(Emphasis added).
Brabb estimated this loss alone at a minimum of 10 kg and a
maximum of 25 kg.   (Id. at p. 5).

[393]   Using Klementiev's ARFs, the court calculates that a
total of 9,524 kg made it to the filters: 1) 70.8 kg yearly in
231-Z Casting over a 23 year period (1,628.4 kg total); 2) 8.6 kg

ORDER RE SUMMARY JUDGMENT-   494

1 Defendants say the plaintiffs are contending MUF documents

2 "show that only 600 kg were lost to all sources," which is

3 sixteen times less the amount of MUF (10,000 kg) necessary to

4 support Klementiev's 20,000 Ci estimate. Furthermore, defendants

5 point out that 10,000 kg in MUF means that nearly 12% of all

6 plutonium processed at Hanford was lost to the ventilation system

7 (10,000 kg of 80,000 kg = 12%). (Klementiev Dep. at pp. 826-

8 27). Klementiev opined that this was "possible" (Id. at p.

9 827), although elsewhere in his deposition he indicated his best

10 estimate of the amount lost to the ventilation system and the

11 stack from "all the sources at PFP" was 0.05 to 1%. (Id. at p.

12 695).

13 The court recognizes that defendants throw out these figures

14 for the first time in their reply brief and thus, plaintiffs have

15 not had an opportunity to explain these apparent contradictions.

16 Although the available documents may only show 600 kg MUF,

17 plaintiffs might contend it is just the tip of the iceberg.

18 Nonetheless, the court must say that 10,000 kg is a staggering

19 amount of plutonium to lose if one assumes a total of 80,000 kg

20 was processed.

21 In the final analysis, the court must agree that at least

22 what is shown by the documents concerning MUF- the 600 kg loss-

23

24

25

_____

26 yearly in 231-Z Burning over a 19 year period (163.4 kg total);
3) 322.14 kg yearly in 234-5Z Processing over a 23 year period
(7,409 kg total); and 4) 62.13 kg yearly in 234-5Z Casting over

27 an 11 year period (683.43 kg total).

28

ORDER RE SUMMARY JUDGMENT-   495

1 does nothing to support Klementiev's 20,000 Ci estimate.[394]

2

3   **(v)   Summary/Daubert Criteria**

4   Klementiev's "process analysis" which produces his 20,000 Ci

5 estimate is not scientifically reliable. The airborne release

6 fractions (ARFs) used as part of that analysis are not supported

7 by the sources upon which Klementiev purports to rely. Secondly,

8 Klementiev's 82% filtration efficiency value, also used as part

9 of his "process analysis," is not supported by the sources upon

10 which Klementiev purports to rely, in particular Dr. Jervis whom

11 the plaintiffs claim is their filtration expert.

12   The scientific unreliability of Klementiev's 20,000 Ci

13 estimate is manifested by his unwillingness to assert that it is

14 his most reliable estimate, as well as his statement that his

15 0.15 Ci estimate, based on his "stack sampling analysis," cannot

16 be ruled out and deserves serious consideration. And while there

17 may be questions about the accuracy and relevancy of certain

18 monitoring data, the failure of plaintiffs' experts, including

19 Klementiev, to even address the data only increases doubt about

20 the thoroughness with which plaintiffs approached the issue of

21 plutonium source term estimation. Likewise, the MUF or B-PID

22 issue offers nothing to support the reliability of Klementiev's

23 20,000 Ci estimate.

24 _____

25   [394] Plaintiffs say certain portions of the Z-Plant exhaust
system have not been assayed. One can only speculate that large
26 amounts of plutonium are to be found in those portions of the
stack. This allegation is insufficient to save Klementiev's
27 20,000 Ci release estimate.
28
    ORDER RE SUMMARY JUDGMENT-   496

1   Klementiev's opinions are not derived from legitimate

2   preexisting research unrelated to this litigation.   Furthermore,

3   his opinions have not been subjected to normal scientific

4   scrutiny through peer review and publication.   There is no

5   indication that the method by which Klementiev derived his ARFs

6   and his filter efficiency values, or the ARFs and the filter

7   efficiency values themselves, are "generally accepted" within the

8   scientific community.   Finally, as noted above, Klementiev fails

9   to show how the method by which he derived his 20,000 Ci estimate

10   is objectively and independently validated by the sources and the

11   data upon which he relied.   Claar v. Burlington Northern

12   Railroad, 29 F.3d 499, 501 (9th Cir. 1994); O'Connor v.

13   Commonwealth Edison Co., 13 F.3d 1090, 1107 (7th Cir. 1994);

14   Muzzey v. Kerr-McGee Chemical Corp., 921 F. Supp. 511, 519 (N.D.

15   Ill. 1996).

16   The court will exclude Klementiev's "process analysis" and

17   the 20,000 Ci estimate because they do not meet the reliability

18   prong of Daubert.   In turn, the court will also exclude the work

19   of plaintiffs' experts Stewart and Crawford-Brown which is based

20   on that estimate.   Plaintiffs do not dispute defendants'

21   assertion that Stewart and Crawford-Brown base their work on

22   Klementiev's estimate.

23

24   **(b)   Qualifications**

25   An individual must be qualified "by knowledge, experience,

26   training or education" to render an opinion on a particular

27   question or subject.   FRE 702.

28

ORDER RE SUMMARY JUDGMENT-   497

1   Defendants assert Klementiev's "process analysis" requires

2 certain expertise which he does not possess. Such expertise

3 relates to airborne release fractions, aerosol physics,

4 industrial processing of metals, ventilation engineering, and

5 HEPA filtration. Defendants note that Klementiev's resume

6 (Defendants' Ex. 173) shows he has spent his career developing

7 computer models related to public health statistics. There is no

8 indication of a background in the engineering analysis of

9 industrial processes, in particular plutonium production

10 processing.

11   At his deposition, Klementiev confirmed that this litigation

12 represented his first foray into radionuclide source term

13 estimation. (Klementiev Dep. at pp. 197-204). Klementiev

14 acknowledged that prior to this litigation, he had never

15 conducted original scientific research regarding how plutonium is

16 released from plutonium manufacturing processes, or how any type

17 of radionuclides are released from any type of manufacturing

18 processes. (Id. at p. 205). The publications listed in his

19 resume do not reveal anything related to radionuclide source term

20 estimation or plutonium processing. Klementiev confirmed as much

21 at his deposition. (Id. at pp. 192-96).

22   However, plaintiffs argue as follows:

23   Without citation to any scientific references,
           defendants make the bare assertion that Klementiev
24   must be an expert in 'airborne release fractions,
           aerosol physics, industrial processing of metals,
25   ventilation engineering, and HEPA filtration,' . .
           . . This is submitted by the same defendants who
26   characterize HEDR's plutonium analysis as 'unassailable
           good science,' even though HEDR never looked
27   at 'airborne release fractions, aerosol physics,
28

ORDER RE SUMMARY JUDGMENT-   498

industrial processing of metals, ventilation
engineering, and HEPA filtration;' and, more to the
point, HEDR never looked at Z plant operations but
merely took some retrospective estimates of combined
separations plant and Z plant plutonium stack releases
that were contained in . . . Anderson at face value
as an excuse to assume that Z plant releases were not
worth calculating.

(Plaintiffs' Response Br. at p. 22).

Plaintiffs ignore the question of Klementiev's

qualifications and attempt to focus attention, once again, on the

purported inadequacies of HEDR. However, the issue here is not

HEDR's analysis of plutonium emissions, but Klementiev's "process

analysis" of plutonium emissions. HEDR did not engage in a

"process analysis" of plutonium emissions. Klementiev did and it

is clear that expertise in airborne release fractions, aerosol

physics, industrial processing of metals, ventilation

engineering, and HEPA filtration is pertinent to such an

analysis. Klementiev's "process analysis" is itself an

acknowledgement that such expertise is necessary. Klementiev

derived his ARFs from Mishima's work in this area. Klementiev

deferred to the expertise of Jervis regarding filtration

efficiencies. Plaintiffs are unable to show that Klementiev has

independent expertise in any of these areas- ARFs, filtration

efficiencies, aerosol physics, industrial processing of metals

and ventilation engineering.

There is some suggestion from plaintiffs that the expertise

of Jervis compensates for any lack of expertise on the part of

Klementiev. First of all, Jervis said nothing about airborne

release fractions and therefore, his opinion cannot salvage that

ORDER RE SUMMARY JUDGMENT-   499

component of Klementiev's "process analysis." The most Jervis could salvage would be Klementiev's 82% filtration efficiency value, but, as discussed above, there is nothing indicating he endorsed that value or the 20,000 Ci release estimate.

Klementiev's lack of qualification to undertake a "process analysis" manifests itself in the methodological unsoundness of his ARFs and his filtration efficiency values. Klementiev turns out to be very similar to Dr. Mayer, another of the plaintiffs' experts. While Klementiev is adept at crunching the numbers provided to him for a "process analysis," he does not have the necessary qualifications in the underlying substantive fields- ARFs, filtration efficiencies, etc. - to know whether those numbers are at all scientifically reliable.

(c) Fit/Relevancy

Defendants contend Klementiev's 20,000 Ci estimate is the only one of his estimates sufficient to produce doses in excess of the doubling doses necessary to raise an inference of causation. The plaintiffs do not respond to that assertion. However, the work done by plaintiffs' expert, Robert Goble, appears to confirm this is the case.[395]

Klementiev's 20,000 Ci estimate is one of the bases for Goble's estimate of organ doses received by individuals residing at Ringold via the "inhalation" or air pathway. Goble provides 99th percentile doses based on Klementiev's analysis, meaning the

_____

[395] Klementiev's other estimates range from 0.15 Ci to 1,168 Ci.

ORDER RE SUMMARY JUDGMENT-   500

1 likelihood somebody actually received such a dose is 1%.

2   Goble actually uses only a portion of Klementiev's 20,000 Ci

3 estimate. Based on Klementiev's "process analysis," Goble

4 calculates that approximately 10,000 Ci alone was released in the

5 period between 1955-65. (Goble 1997 Report at p. 18).[396] From

6 this 10,000 Ci, Goble arrives at mean, 90th percentile, 95th

7 percentile, and 99th percentile doses for "adult individuals

8 residing at Ringold for the period 1955-65." (See Table 3 at p.

9 6, Goble Declaration, Ex. 2 to Plaintiffs' Non-Iodine Appendix

10 1).

11   Exclusion of Klementiev's 20,000 Ci estimate obviously

12 results in exclusion of these doses. To the extent other of the

13 plaintiffs' experts rely on this estimate for concentration,

14 deposition, and dose estimates, their opinions must likewise be

15 excluded.

16

17   **b.   Robert Goble/Surviving Non-Thyroid Cancer Claims**

18   Dr. Goble is a Research Professor of Environment,

19 Technology, and Society, and Adjunct Professor of Physics at

20 Clark University in Worcester, Massachusetts. He has a Ph.D. in

21 physics.

22   Goble's initial non-iodine expert report (March 1996)[397]

23 ───────────────────

   [396] Defendants observe that Goble's estimate that 1 to 2% of
24 the MUF was released to the atmosphere (Goble Dep. at pp. 368-70)
   is inconsistent with a 10,000 Ci (approximately 160 kg) release
25 between 1955-65. The total MUF reported for 1956-66 was 600 kg.
   1 to 2% of 600 kg amounts to 6 to 12 kg (370 to 735 curies).

26
   [397] "Estimating Exposures from Releases of Radioactive
27 Elements Other than Iodine at Hanford."
28
   ORDER RE SUMMARY JUDGMENT-   501

1 and his supplemental report (March 1997)[398] provide single

2 point-estimates (mean estimates) of non-iodine dose. Goble's

3 dose estimation method involves adjusting (increasing) the dose

4 output of the HEDR non-iodine spreadsheets based on a number of

5 different multiplication factors.[399]

6   Defendants' expert, Dr. John R. Frazier, computed "maximum

7 hypothetical doses" that could result from Goble's method.

8 (Frazier June 1997 Affidavit, Defendants' Ex. 162). Frazier did

9 this for the nine locations covered by HEDR's spreadsheets,

10 including Ringold.[400] Ringold was the maximum **non-iodine** dose

11 location with a cumulative Effective Dose Equivalent dose[401] of

12 3,667 millirem (3.7 rem) for the entire release period of 1944 to

13 1987. (Table 3 of Frazier June 1997 Affidavit). Of the HEDR

14 locations, Ringold is the closest to the Hanford facility.

15   Frazier then converted his EDE for Ringold into organ doses

16 using the conversion factors in ICRP (International Commission on

17 Radiological Protection) 56 and Goble's approach. These organ

18 _____

19 [398] "Estimating Exposures from Releases of Plutonium at
Hanford: Supplementary Report."

20 [399] According to defendants, Goble's increase in plutonium
doses is due to his disagreement with HEDR over the amount of
21 plutonium released to the atmosphere. Whereas HEDR estimates the
separations plants released 1.78 Ci of plutonium between 1944 and
22 1972, Goble estimates a total release of 2,170 Ci (34.72 kg) from
the separations plants, the PFP (Z-Plant) and the resuspension of
23 buried plutonium due to the burrowing activities of gophers at
Hanford waste sites.

24
[400] The nine locations are: Eltopia, Lewiston, Pendleton,
25 Richland, Ritzville, Spokane, Sunnyside, Wenatchee, and Ringold.

26 [401] EDE is "a quantity that is used to express a total dose
to the body based on the doses to the various individual organs
27 of the body." (Frazier 1997 Affidavit at p. 7, paragraph 19).
28

ORDER RE SUMMARY JUDGMENT-   502

doses are found at Table 13 of Frazier's June 1997 affidavit. For each organ, Frazier calculated the cumulative dose from each of the following elements: strontium-90, ruthenium-103, ruthenium-106, cerium-144, cesium-137, iodine-131 and plutonium-239. These doses were then added together to provide a cumulative organ dose from all elements. For example, the cumulative organ dose for the liver is 7,047 millirem (7 rem), of which Pu-239 contributes 6,893 millirem (6.9 rem).[402]

In their opening non-iodine brief, defendants compared the cumulative organ doses calculated by Dr. Frazier to the doubling doses for each cancer site (i.e. organ site) as derived from Dr. Radford's risk co-efficients. In each case, the organ dose received was less than the doubling dose. (See Table at p. 32 of Defendant's Opening Non-Iodine Brief). On this basis, defendants sought dismissal of **all** non-thyroid cancer claims.

In response to defendants' Motion for Summary Judgment Re Non-Iodine and the accompanying affidavit of Dr. Frazier, the plaintiffs submitted a November 1997 declaration from Dr. Goble. (Ex. 2 to Plaintiffs' Appendix I re Non-Iodine Claims). This declaration, unlike his reports, provides a range of organ doses (a "probability distribution") including 90th, 95th and 99th percentile doses. Of the non-iodine elements, Goble's declaration provides only **plutonium** organ doses.

The plutonium organ doses found in Frazier's Table 13 are

---

[402]   Insofar as the non-iodine elements, Pu-239 constitutes the vast majority of the cumulative dose for each organ site. (Table 13 of Frazier Affidavit).

ORDER RE SUMMARY JUDGMENT-   503

1   those listed by Goble in his November 1997 declaration as his

2   "mean" estimates. For example, Frazier's Table 13 lists a

3   cumulative plutonium dose to the bone of 31,563 millirem. In

4   Table 1 of his declaration, Goble lists his "mean" dose to the

5   bone as 31.6 rem. Frazier's Table 13 lists a cumulative

6   plutonium dose to the lungs of 11,610 millirem. In Table 1 of

7   his declaration, Goble lists his "mean" dose to the lungs as 11.6

8   rem. Goble agreed there was **"mutual understanding"** between he

9   and Frazier as to the "methods of calculation." (Goble November

10   1997 Declaration at p. 1)(Emphasis added). Goble's "mean"

11   plutonium doses are 73rd percentile doses. Therefore, it is 27%

12   likely that an individual residing continuously at Ringold

13   between 1944 to 1987 received such a dose. (Frazier January 1998

14   Affidavit at pp. 3-5, Defendants' Ex. 215).

15   Goble's "probability distribution" of doses is displayed in

16   plaintiffs' "Table II: Dr. Goble's Hypothetical Organ Doses for

17   Highly Exposed Individuals Compared with Defendants' Estimates,"

18   attached to their "Joint Response to Defendants' Motion for

19   Summary Judgment Re Non-Iodine." Table II shows Goble's

20   estimated organ doses for over twenty different organs or cancer

21   sites based on three different pathways: the plutonium

22   inhalation pathway; the iodine-131 backyard cow pathway; and the

23   river pathway.

24   Two sets of doses are provided for the plutonium inhalation

25   pathway. These are 99th percentile doses meaning there is a 1%

26   likelihood that any individual residing continuously in Ringold

27   between 1944-1987 received such a dose. One set of doses is

28   **ORDER RE SUMMARY JUDGMENT-   504**

1 based on the plutonium source term analysis of plaintiffs'

2 expert, Dr. Thomas Cochran. The other set is based on the

3 plutonium source term analysis of plaintiffs' expert, Dr.

4 Alexandre Klementiev. The doses derived from Klementiev's source

5 term analysis are four to five times higher than the doses

6 derived from Cochran's source term analysis. As indicated above,

7 the court is excluding Klementiev's source term analysis on

8 Daubert grounds.

9   Dr. Goble also calculated 95th percentile iodine-131 doses

10 for adults and children living in Ringold who drank milk from a

11 backyard cow in 1945, the peak year for iodine-131 emissions. A

12 95th percentile dose means there is a 5% likelihood that an adult

13 or child living in Ringold received this dose by drinking milk

14 from a backyard cow in 1945. Dr. Goble's I-131 doses are

15 provided for a limited number of cancers: parathyroid, salivary

16 gland, stomach, small intestine, bladder and breast (lactating

17 female).[403] For example, Table II shows an adult dose of 39 rem

18 and a child dose of 86 rem for salivary gland cancer.

19   Finally, Dr. Goble calculated 90th percentile radiation

20 doses based on Dr. Hattis' analysis of Columbia River emissions.

21 The 90th percentile dose means there is a 10% likelihood someone

22 residing in Ringold received this dose based on his/her

23

24

25

---

26   [403] Goble's doses are derived from risk co-efficients and
doses provided by plaintiffs' expert Dr. Finston for these

27 particular cancer sites. (Goble 1997 Declaration at pp. 6-7).

28 **ORDER RE SUMMARY JUDGMENT-   505**

1 consumption of 90 pounds of fish per year between 1945 and

2 1987.[404] There are two sets of doses. The first set of river

3 doses (the lower doses), are based on HEDR's assumptions about

4 the BCF (bioconcentration factor) in fish using the **mean** numbers

5 proposed by Hattis in his original 1996 report (population risk

6 analysis), rather than HEDR's **median** numbers. The second set of

7 river doses (the higher doses) are derived from the analysis

8 found in Hattis' 1997 supplemental report assuming the

9 "possibility" of a BCF of 66,700 for P-32 for **all** fish in the

10 Columbia River. For example, with regard to colon cancer the

11 lower dose is 12 rem and the higher dose is 1,240 rem.[405] As

12 indicated above, the court is excluding Hattis' river analysis on

13 <u>Daubert</u> grounds.

14   Next to each cancer site listed on plaintiffs' Table II are

15 Dr. Radford's co-efficients of excess relative risk. Two sets of

16 co-efficients are provided. The lower figure is the co-efficient

17 _____

18   [404] HEDR assumed its "maximum representative individual"
   consumed 90 pounds of resident fish on an annual basis. In his
19 supplemental report and affidavit, Hattis provides organ doses
   for such "maximum representative individuals" at various
20 locations, including Ringold. Goble's 90th percentile doses are
   derived from Hattis' organ doses for the "maximum representative
21 individual" at Ringold.

22   [405] It is not exactly clear to the court how Goble came up
   with these 90th percentile doses. However, looking at the organ
23 dose tables in Hattis' affidavit, and specifically the doses for
   the "maximum representative individual" at Ringold, it appears
24 Goble's 90th percentile doses are approximately double Hattis'
   doses. For example, Hattis reports a colon or lower large
25 intestine dose of 14 rem for the "maximum representative
   individual" at Ringold "after mean/median correction" and 616 rem
26 "after increase in 32P BCF to 66,700 for all fish." (Table FF in
   Hattis Affidavit). Goble's 90th percentile doses are 28 rem and
27 1240 rem.

28 **ORDER RE SUMMARY JUDGMENT-   506**

1 without an increased susceptibility factor. The higher figure is

2 the co-efficient including Radford's five-fold increased

3 susceptibility factor. Table II provides "Doubling Doses

4 Corrected For Susceptibility" which are calculated from Radford's

5 co-efficients incorporating his increased susceptibility

6 factor.[406]

7   For example, as concerns bone cancer, the co-efficient

8 without the increased susceptibility factor is 0.60. With the

9 susceptibility factor it is 3.00 (0.60 x 5). A risk co-efficient

10 of 3.00 produces a doubling dose of 33 rem (33,000 millirem). A

11 risk co-efficient of 0.60 produces a doubling dose of 167 rem

12 (167,000 millirem), approximately five times more than 33 rem

13 (33,000 millirem). Radford's susceptibility factor is

14 scientifically unreliable. Accordingly, the court concerns

15 itself only with risk-coefficients and doubling doses which do

16 not incorporate the increased susceptibility factor.[407]

17   Because the court is excluding Klementiev's plutonium source

18 term analysis, it need not consider the 99th percentile plutonium

19

20

21 _____

    [406] Next to the column showing "Doubling Doses Corrected for
22 Susceptibility" is a column labeled "Estimated 'Maximum' Organ
    Doses." These are Dr. Frazier's cumulative organ doses from "all
23 air pathway radionuclides" (Sr-90, Ru-103, Ru-106, I-131, Cs-137,
    Ce-144, and Pu-239) found in Table 13 of his affidavit. For
24 example, Frazier's cumulative organ dose for bone surfaces is
    32,699 millirem which is displayed in plaintiffs' Table II as a
25 rounded off figure of 33 rem.

26   [407] These risk co-efficients and doubling doses are listed
    supra in the section regarding "Non-Iodine Exposures" and "Health
27 Effects."
28
    ORDER RE SUMMARY JUDGMENT–   507

1 inhalation doses Goble derives from that analysis.[408] That

2 leaves the 99th percentile plutonium inhalation doses Goble

3 derives from Cochran's plutonium source term analysis.[409]

4   Because the court is excluding Hattis' river analysis, it

5 need not consider the 90th percentile doses Goble derives from

6 that analysis.[410] **Assuming** HEDR's river dosimetry is

7 scientifically reliable, that leaves it for the purpose of

8 computing river doses. However, in each case, doses derived from

9 HEDR using its **median** numbers regarding BCF would be even less

10 than the 90th percentile doses derived by using its mean numbers

11 as proposed by Hattis. In other words, HEDR's doses for an

12 individual living in Ringold and annually consuming 90 pounds of

13 resident fish between 1945 and 1987 would be less than the 90th

14 percentile doses derived from using HEDR's mean numbers as

15

16 _____

17   [408] The 99th percentile doses derived from Klementiev's
    source term analysis exceed the applicable doubling doses for
18 only four cancers: bone, lung, leukemia, and liver.

19   [409] HEDR's doses, of course, would be even lower.

20   [410] For the following cancer sites found on plaintiffs'
    Table II, the only dose which exceeds the applicable doubling
21 dose is Hattis' river dose based on a 66,700 BCF for all fish:
    colon, rectum, non-Hodgkins' lymphoma, small intestine, breast
22 (lactating female and non-lactating female), ovary, testes,
    bladder, brain and nervous system, and skin (non-melanoma).
23   Defendants assert yet another reason for disregarding the
    66,700 BCF is because it is the value for **stable (non-**
24 **radioactive) phosphorous** as opposed to radioactive phosphorous.
    In support, defendants have submitted the affidavit of their
25 expert, Dr. Frazier (Defendants' Ex. 210). It appears this
    argument is tendered for the first time in defendants' reply
26 submission. Therefore, the plaintiffs have not had an
    opportunity to respond to it. However, with or without this
27 argument, plaintiffs' river case must be dismissed.
28
    ORDER RE SUMMARY JUDGMENT-   508

1 proposed by Hattis.[411] For each cancer site listed on Table II,

2 these 90th percentile doses (the lower doses) are significantly

3 less than the applicable doubling doses for each cancer site.

4   To the extent I-131 contributes to cumulative organ dose of

5 an individual who resided in Ringold, it is recognized exposure

6 would not necessarily be limited solely to 1945 and solely from a

7 backyard cow. Although I-131 emissions may have been greatest in

8 1945, emissions obviously occurred in subsequent years in lesser

9 amounts. Although milk from a backyard cow fed on pasture grass

10 may constitute the greatest source of ingested I-131, there are

11 other milk pathways and other pathways in general which could

12 constitute a source of exposure.

13   Considering all of this, the following is a comparison of

14 Goble's doses with the applicable doubling doses for each cancer

15 site:

16

17   1) Cancer of Nasal Cavity: The doubling dose is 455 rem.

18 Goble's 99th percentile plutonium inhalation dose (based on

19 Cochran source term analysis) is 42 rem.

20

21   2) Cancer of the Oral Cavity and Pharynx: 345 rem is the

22 applicable doubling dose. Goble's 99th percentile plutonium

23 inhalation dose (based on the Cochran source term analysis) is 42

24 rem.

25 _____

[411] For example, Goble's 90th percentile dose for colon
26 cancer based on HEDR's BCF assumption and using its mean numbers,
    as proposed by Hattis, is 28 rads. HEDR's dose would be less
27 because of the use of median numbers.

28 **ORDER RE SUMMARY JUDGMENT-   509**

3) Pancreatic Cancer: 556 rem is the applicable doubling dose. Goble's 99th percentile plutonium inhalation dose (based on Cochran source term analysis) is 0.1 rem.

4) Colon Cancer: 139 rem is the applicable doubling dose. Goble's 99th percentile plutonium inhalation dose (based on the Cochran source term analysis) is 0.1 rem.

5) Non-Hodgkin's Lymphoma (Males Only): 167 rem is the applicable doubling dose. Goble's 99th percentile plutonium inhalation dose (based on the Cochran source term analysis) is 12 rem.

6) Esophageal Cancer: 400 rem for males is the applicable doubling dose; 286 rem for females ages 21 and over is the applicable doubling dose. Goble's 99th percentile plutonium inhalation dose (based on the Cochran source term analysis) is 0.8 rem. (See Table II of Goble November 1997 Declaration at p. 5).

7) Ovarian Cancer: 101 rem is the applicable doubling dose. Goble's 99th percentile plutonium inhalation dose (based on the Cochran source term analysis) is 3.3 rem.

8) Testicular Cancer: 29 rem is the applicable doubling dose. Goble's 99th percentile plutonium inhalation dose (based on the Cochran source term analysis) is 3.3 rem.

ORDER RE SUMMARY JUDGMENT-   510

1   9) Rectal Cancer: 476 rem is the applicable doubling dose.

2 Goble's 99th percentile plutonium inhalation dose (based on the

3 Cochran source term analysis) is 0.1 rem.

4

5   10) Stomach Cancer: 313 rem is the applicable doubling

6 dose. Goble's 99th percentile plutonium inhalation dose (based

7 on the Cochran source term analysis) is 0.1 rem.

8   Goble provides a 95th percentile I-131 dose of 1.1 rem for

9 an adult and 6.3 rem for a child residing in Ringold and drinking

10 milk from a backyard cow in 1945. Even making the unreasonable

11 assumption that the same doses would continue to be received over

12 an additional 41 year period (1946-87)[412], the cumulative doses

13 would still not surpass the doubling dose (1.1 x 41 = 45.1 rem;

14 6.3 x 41 = 258.3). For the stomach wall, Frazier's cumulative I-

15 131 dose for the maximally exposed Ringold adult (as derived from

16 Goble's dose estimation method) is 297 millirem (0.297 rem).

17 (Table 13 to Frazier Affidavit).

18

19   11) Salivary Gland Cancer: Applicable doubling doses are 33

20 rem for adults (ages 20 and over); 17 rem for children (ages 10-

21 19); 10 rem for infants (ages 0-9). Goble's 99th percentile

22 plutonium inhalation dose (based on Cochran source term analysis)

23 is 0.1 rem.

24   Goble's 95th percentile I-131 doses are 39 rem for an adult

25 _____

26   [412] "Unreasonable" because of decreasing releases of I-131
to the atmosphere and the fact that a 41 year period would likely
27 encompass both adult and child doses.

28

ORDER RE SUMMARY JUDGMENT-   511

1 and 86 rem for a child. **These doses exceed the applicable**

2 **doubling doses.** This is discussed <u>infra</u>.

3

4   12) Cancer of the Urinary Tract and Kidneys: 81 rem is the

5 applicable doubling dose (100 rem for bladder; 140 rem for

6 kidney). Goble's 99th percentile plutonium inhalation dose for

7 the bladder (based on the Cochran source term analysis) is 0.1

8 rem and for the kidney 0.9 rem. Combined dose is 1.0 rem.

9   For the bladder, Goble provides 95th percentile I-131 doses

10 of 1.1 rem for a Ringold adult and 6.3 rem for a Ringold child

11 consuming milk from a backyard cow. Again, making the

12 unreasonable assumption that the same doses would continue to be

13 received over an additional 41 year period (1946-87), the

14 cumulative doses would still not surpass the doubling dose (1.1 x

15 41 = 45.1 rem; 6.3 x 41 = 258.3 rem). For the bladder wall,

16 Frazier's cumulative I-131 dose for the maximally exposed Ringold

17 adult (as derived from Goble's dose estimation method) is 38

18 millirem (0.038 rem). (Table 13 to Frazier Affidavit). Goble

19 does not provide an I-131 dose for the kidneys.

20

21   13) Bone Cancer: 167 rem is the applicable doubling dose.

22 Goble's 99th percentile plutonium inhalation dose (based on the

23 Cochran source term analysis) is 258 rem which **exceeds the**

24 **applicable doubling dose.**[413] This is discussed <u>infra</u>.

25

26 _____

[413] Goble's 95th, 90th and 73rd percentile doses are all

27 below 167 rem.

28 **ORDER RE SUMMARY JUDGMENT-   512**

14) Lung Cancer: 77 rem is the applicable doubling dose for non-smokers exposed at age 10 or over; 250 rem is the applicable doubling dose for smokers exposed at age 10 or over. Goble's 99th percentile plutonium inhalation dose (based on the Cochran source term analysis) is 95 rem. **95 rem exceeds the applicable doubling dose for non-smokers exposed at age 10 and over.**[414] This is discussed <u>infra</u>.

15) Liver Cancer: 204 rem is the applicable doubling dose for individuals exposed at ages 10 or over, with the exception of females ages 10-19 at the time of exposure for whom the doubling dose is 588 rem. Goble's 99th percentile plutonium inhalation dose (based on the Cochran source term analysis) is 56 rem.

16) Leukemia (excluding chronic lymphocytic variety): 22 rem is the applicable doubling dose. Goble's 99th percentile plutonium inhalation dose (based on Cochran source term analysis) is 12 rem.

17) Cancer of the Nervous System and the Brain: 31 rem is the applicable doubling dose. Goble's 99th percentile plutonium inhalation dose (based on Cochran source term analysis) is 0.1 rem.

18) Breast Cancer (Lactating and Non-Lactating Females):

---

[414] Goble's 95th, 90th and 73rd percentile doses are all lower than the 77 rem doubling dose for non-smokers.

ORDER RE SUMMARY JUDGMENT-   513

1   63 rem is the applicable doubling dose. Goble's 99th percentile

2   plutonium inhalation dose (based on Cochran source term analysis)

3   is 0.1 rem.

4   Goble provides a 95th percentile I-131 dose of 14 rem for a

5   lactating adult female residing in Ringold who consumed milk from

6   a backyard cow in 1945. This does not exceed the applicable

7   doubling dose, but additional exposures in subsequent years

8   during additional lactation periods could increase the cumulative

9   exposure above the doubling dose. This is discussed _infra_.

10

11   19) Non-Melanoma Skin Cancer: 100 rem is the applicable

12   doubling dose. Goble's 99th percentile plutonium inhalation dose

13   (based on Cochran source term analysis) is 0.1 rem.

14

15   20) Prostate Cancer: 345 rem is the applicable doubling

16   dose. Presumably falls into Goble's "other" category and

17   therefore, 99th percentile plutonium inhalation dose (based on

18   Cochran source term analysis) is less than 0.2 rem. (Goble

19   November 1997 Declaration, Tables I and II, pp. 4-5).

20

21   21) Gallbladder Cancer: 833 rem is the applicable doubling

22   dose. Presumably falls into Goble's "other" category and

23   therefore, 99th percentile plutonium inhalation dose (based on

24   Cochran source term analysis) is less than 0.2 rem. (Goble

25   November 1997 Declaration, Tables I and II, pp. 4-5).

26

27   Because plaintiffs' Table II employs the maximizing

28   **ORDER RE SUMMARY JUDGMENT-   514**

1 assumptions regarding non-iodine exposure (99th percentile doses;

2 the maximum dose location (Ringold); the maximum number years of

3 exposure (1944 to 1987); the maximum representative individual in

4 terms of river exposure), the court finds it appropriate to

5 dismiss the majority of plaintiffs' non-thyroid cancer claims,

6 including: colon, rectum, leukemia, non-Hodgkin's lymphoma,

7 liver, nasal cavity, oral cavity and pharynx, stomach, ovary

8 testes, bladder, kidney, pancreas, brain and nervous system,

9 breast (non-lactating female); skin (non-melanoma), prostate and

10 gallbladder. Based on plaintiffs' Table II and Frazier's Table

11 13, it is not apparent how the maximally exposed individual at

12 Ringold, and therefore any plaintiff for that matter, could have

13 received even a combination of doses from plutonium, other non-

14 iodine radionuclides including those in the river, and

15 radioiodine (in the appropriate case)[415], exceeding any of the

16 doubling doses for these cancers.

17   Plaintiffs' Table II lists parathyroid cancer, small

18 intestine cancer, spleen cancer, and thymus cancer. The court

19 cannot find where Radford makes mention of any of these cancers

20 in his non-iodine report or his deposition testimony. Indeed,

21 defendants assert no plaintiff has filed a claim for parathyroid

22 cancer. The risk co-efficient used in Table II for each of these

23 cancers is the 0.63 ERR/Sv figure (without an increase for

24 Radford's susceptibility factor).

25 ───────────────

    [415] Frazier's cumulative I-131 organ doses (based on Goble's
26 dose estimation method) are very low, with the exception of the
    cumulative thyroid dose (435 rem). (Table 13 to Frazier
27 Affidavit, Defendants' Ex. 162).

28
    ORDER RE SUMMARY JUDGMENT-   515

1   In his post-deposition declaration dated November 1997,

2   Radford cited the Pierce study and asserted it was proper to use

3   this figure as a risk co-efficient for all solid tumors for which

4   a "statistically significant" excess has not been found (so-

5   called "rare" cancers). As already discussed, the court will not

6   allow use of that risk co-efficient for prostate cancer, non-

7   Hodgkin's lymphoma, gallbladder cancer, nasal cavity cancer,

8   esophageal cancer, and cancer of the oral cavity and pharynx.

9   This is because Radford brought the figure up for the first time

10 in his declaration, after both his report and deposition had been

11 completed. For the same reason, the court will exclude its use

12 for any other types of cancer.

13   Furthermore, even in his declaration, Radford says nothing

14 about parathyroid cancer, small intestine cancer, spleen cancer,

15 or thymus cancer. Therefore, assuming any such claims exist,

16 they will be dismissed with prejudice for lack of expert

17 proof.[416] Goble's declaration, submitted after the expert

18 report deadline, is insufficient to sustain any such claims.

19 //

20 //

21 _____

        [416] In any event, small intestine cancer would be subject to
22 the same fate as stomach cancer claims. The figures are
    identical in terms of 99th percentile plutonium inhalation doses
23 and 95th percentile backyard cow I-131 doses. Those figures are
    not even sufficient to exceed the applicable doubling dose (160
24 rem) using an ERR/Sv of 0.63. For the spleen and thymus, the
    99th percentile plutonium inhalation doses are minuscule and come
25 nowhere close to exceeding the applicable doubling dose (160 rem)
    using an ERR/Sv of 0.63. Even a dose contribution from other
26 non-iodine radionuclides, including those found in the river, and
    an iodine dose (insofar as the small intestine) could not sustain
27 these claims.
28
    ORDER RE SUMMARY JUDGMENT-   516

(1) Bone and Lung Cancer

In only two cases do Goble's 99th percentile plutonium inhalation doses (based on Cochran source term analysis) exceed the applicable doubling dose: 1) Bone Cancer; and 2) Lung Cancer for non-smokers ages 10 or over at the time of exposure.

Defendants contend that even so, the court should dismiss **all** non-thyroid cancer claims. According to defendants, Goble should be bound by the mean (73rd percentile) doses found in his non-iodine reports. If that were the case, the bone cancer and non-smoker lung cancer claims would indeed require dismissal. The mean dose for bone cancer is 32 rem, well below the 167 rem doubling dose. The mean dose for lung cancer is 12 rem, well below the 77 rem doubling dose for non-smokers.

The defendants contend there are four reasons why Goble should not be allowed to use 99th percentile doses. First, defendants cite Goble's April 1996 medical monitoring report wherein he states:

> In the case of generic causation confronting
> the 'question whether we are making overestimates
> or underestimates of exposure' in attempting to
> assess causation is relatively straightforward.
> **The object is to present a mean or 'best estimate.'**
> Uncertainties should be described and degrees
> of confidence assessed in order to provide others
> a perspective in interpreting the exposure
> estimates.

(Goble April 22, 1996 Rpt. at p. 11)(Emphasis added).

Secondly, defendants argue the dose estimation approach described in Goble's **reports** generates only a "mean" estimate and at no time has Goble indicated he would use 99th percentile doses for any purposes.

ORDER RE SUMMARY JUDGMENT-   517

1   Thirdly, defendants argue the plaintiffs have no way of

2 determining whether an individual has characteristics that would

3 result in the 99th percentile dose because, for example, Goble

4 does not identify any means for determining which claimants were

5 exposed to doses higher than his "mean" doses. Furthermore,

6 defendants say Goble does not offer any scientific basis for

7 quantifying the extent to which a person's characteristics differ

8 from those of an "average" member of the population.

9   Finally, defendants contend Goble does not "pretend" to

10 claim that his 99th percentile estimate applies to any plaintiff

11 in this case.

12   In sum, defendants argue it is too late for Goble to change

13 his dose estimation approach and that based on his **reports** he is

14 bound to use a value no higher than his "mean" estimates and

15 compute doses using only those parameters and assumptions which

16 were timely disclosed in those **reports** (i.e. in accord with the

17 court's scheduling orders).

18   Defendants did not file a formal motion in limine seeking

19 exclusion of Goble's dose estimation method. Consequently,

20 although defendants allude to deficiencies in Goble's

21 methodology, those matters are not formally before the court.

22 For example, in a footnote, defendants assert the plutonium

23 release estimate used by Goble (Cochran's release estimate)-

24 2,170 Ci (34.72 kg)- cannot be squared with any of the monitoring

25

26

27

28
ORDER RE SUMMARY JUDGMENT-   518

1 data. (Defendants' Non-Iodine Reply Brief Part II, p.27, n.

2 33).[417]

3   Defendants say the reason they did not file a motion in

4 limine with regard to Goble is because they figured the maximum

5 doses derived from his methodology were so low they would not

6 exceed any of the applicable doubling doses. (Ftn. 22 at p. 33

7 of Defendants' Reply Brief). That is most definitely true with

8 regard to Goble's "mean" or 73rd percentile doses. It is also

9 true, as pointed out, with regard to all of his 99th percentile

10 plutonium inhalation doses with two exceptions: 1) bone cancer;

11 and 2) lung cancer in non-smokers who are exposed at ages 10 or

12 over.

13   Nowhere do the plaintiffs suggest that Goble's expert

14 reports discuss 99th percentile doses. In its review of Goble's

15 reports (March 1996 and March 1997), the court fails to see any

16 specific reference to 99th percentile doses. On the other hand,

17 even Dr. Frazier, defendants' expert, acknowledges Goble

18 discussed "variability" in his March 1997 supplemental report,

19 although he did not "quantify the range of doses implied by this

20 variability." (Frazier January 1998 Affidavit, Defendants' Ex.

21 210, at p. 3).

22   Indeed, Goble devoted an entire section of his March 1997

23 _____

24 [417] In his June 1997 affidavit, Frazier contends there are
   "significant flaws" in Goble's method. (Frazier 1997 Affidavit
   at p. 2, n. 1). In his January 1998 affidavit, Frazier takes
25 issue with Goble's assumptions regarding variability in doses and
   also with plaintiffs' "speculative" plutonium release estimates
26 and their ignoring of "contrary measurements of plutonium at
   Hanford." (Frazier 1998 Affidavit, Defendants' Ex. 210, at pp. 3
27 and 8).
28
   ORDER RE SUMMARY JUDGMENT—   519

1   supplemental report to "Uncertainty and Interindividual

2   Variability in Dose Estimates."   (Section 5.0 at pp. 18 and 19).

3   Goble stated:

>   As discussed in my iodine reports, estimates of
>   variability and uncertainty should accompany
>   dose estimates and it is important to distinguish
>   between them. As indicated in my April report on
>   exposures from non-iodine releases . . . the largest
>   uncertainties in exposures from plutonium stem
>   from the release estimates. Uncertainties in
>   modeling and dosimetry are similar to the case of
>   iodine for the inhalation pathway and have been
>   treated in the HEDR analysis. The new
>   source term analyses of Cochran and Klementiev
>   now permit a quantitative treatment of the release
>   uncertainties and these can be combined with the
>   modeling and dosimetry uncertainties. For Cochran's
>   release estimates, I believe it is reasonable to
>   represent the uncertainty in terms of a log normal
>   distribution . . . .   There are three major sources of
>   variability in the doses received by individuals:
>   i) there will be differences in internal processing
>   of plutonium, its transport to various organs, and
>   the doses delivered by the plutonium to those organs
>   for the same amount of plutonium inhaled; ii) there
>   will be differences in the amount of plutonium in-
>   haled for the same exposure to contaminated air;
>   iii) there will be differences in the actual aggregated
>   exposures to contaminated air, for the same estimates
>   of external concentrations.
>
>   I discuss variability in these sources in my April
>   reports . . . and recommend the use of log normal
>   distributions to characterize the variability. The
>   values for the natural log of geometric standard
>   deviations of the distribution, ln gsd, were 0.7
>   for i) [inhalation dose factor], 0.5 for ii) [breathing
>   rate], and 0.5 for iii) [local differences to be
>   expected in the air concentrations]. Studies . . .
>   bearing on i) which have become available since I
>   wrote those reports suggest that my estimated
>   variability for i) [inhalation dose factor] may
>   have been somewhat low, and I now recommend a choice
>   of 1.0 as an appropriate estimate of the ln gsd.

25   In his November 1997 declaration, Goble used these

26   variability assumptions to compute his 99th, 95th and 90th

27   percentile doses. Tables 1, 2 and 3 (pp. 4-6 of the declaration)

28   **ORDER RE SUMMARY JUDGMENT-   520**

are entitled "Estimated plutonium doses to adult individuals residing at Ringold for the entire release period **illustrating interindividual variability in dose.**" (Emphasis added).

In his 1998 affidavit, Frazier asserts that "Goble does not provide any disciplined analysis of these assumptions regarding variability" and "provides little information about his dose probability distribution or the scientific basis for his variability assumptions." (Frazier January 1998 Affidavit at p. 3, Paragraphs 13 and 14). However, Frazier does not go into detail about this and, as noted above, defendants did not file a Daubert motion against Goble's plutonium dose estimation method. Frazier acknowledges that based on the "ln gsds" provided by Goble, he was able to derive the probability distribution referred to by Goble. In other words, Frazier was able to figure out Goble's median (50th percentile), mean (73rd percentile), 90th, 95th and 99th percentile doses. (Id. at pp. 4-8).

Defendants argue that plaintiffs have no way of determining whether an individual has characteristics that would result in the 99th percentile doses reported by Goble. This problem apparently arises from the fact that HEDR only calculates "average" doses. As pointed out by Frazier, Goble does not change HEDR's methodology, but merely applies adjustment factors. While the median (50th percentile) or the mean (73rd percentile) doses might qualify as "average," the defendants suggest the 99th percentile doses do not. Apparently, defendants are willing to accept that an individual plaintiff who resided continuously in Ringold from 1944-87 qualifies as an "average" person who could

ORDER RE SUMMARY JUDGMENT-   521

1 have received one of Goble's mean or median doses. However, they

2 are not willing to accept that for a 99th percentile dose because

3 they say Goble has not provided any means for distinguishing the

4 "average" individual from the 99th percentile individual.

5   The plaintiffs contend Goble has provided the tools for

6 distinguishing the "average" individual and these are: 1)

7 differences in air concentration because of geographic

8 location[418]; 2) uptake of radionuclides into the body- i.e.

9 breathing rate, breathing capacity, size, weight, time spent

10 outdoors, activity level while outdoors; and 3) inhalation dose

11 factor.[419] In his 1997 supplemental report, Goble provided

12 values for each of these which were later employed by him in

13 calculating 90th, 95th and 99th percentile doses. Frazier

14 confirmed that use of those values would result in the 90th, 95th

15 and 99th percentile doses reported by Goble.

16   In the absence of a <u>Daubert</u> motion which convinces the court

17 that Goble's dose estimation method is scientifically unreliable,

18 the only possible basis for ignoring Goble's 99th percentile

19 doses is his failure to specifically mention them in his expert

20 **reports**. The court is not convinced this is a sufficient basis

21 because in his 1997 supplemental report, Goble clearly laid the

22 _____

23 [418] Plaintiffs say that as a result of processes like "wet
deposition" and/or the effects of high altitude on dose,

24 individual plaintiffs living in areas of high fog, or upon land
at a particular altitude, will be exposed to higher levels of

25 radionuclides in their air than persons living in "average"
terrain in the same sector.

26

27 [419] Among the variables here, according to plaintiffs, is
"organ size."

28

ORDER RE SUMMARY JUDGMENT-   522

1   groundwork for the variability analysis from which he later, in

2   his declaration, calculated his 99th percentile doses.

3   The court will allow bone cancer claims and lung cancer

4   claims, based on plutonium exposure, to proceed into Phase III

5   individual causation discovery.[420]   As a practical matter,

6   however, the assumption underlying Goble's 99th percentile doses-

7   **that the individual resided continuously in Ringold from 1944 to**

8   **1987-** suggests there will be few, if any, individuals, who can

9   prove exposure to a dose in excess of the doubling doses.   Of

10   course, for lung cancer there is the additional qualification

11   that the individual be a non-smoker whose exposure occurred at

12   age 10 or over.[421]

13   Plaintiffs contend that due to "interindividual" variations,

14   persons who live further away from the Hanford plant than Ringold

15   "could easily have experienced higher doses than a person in

16   Ringold."   Plaintiffs also contend that among their ranks are

17   "dozens of [individuals] who lived at what was known as Camp

18   Hanford, or actually on the grounds of the Hanford Reservation,"

19   _____

20   [420] According to Radford, the "principal health effects of
     inhalation of plutonium are from insoluble particles deposited in
21   the bronchial epithelium of the lungs, as well as liver and bone
     cancers from deposition in the body."   (Radford 1996 Non-Iodine
22   Rpt. at pp. 3-4).

23   [421] Note the likelihood there are such plaintiffs is made
     more remote by the fact the individual must have been diagnosed
24   with the condition **after** 1987.   If he is diagnosed with cancer
     before 1987, the years of exposure after the date of diagnosis
25   are irrelevant.   In other words, the individual cannot meet the
     criterion that he be continuously exposed between 1944 and 1987.
26   In general, plaintiffs must show exposures in excess of the
     doubling doses occurred before the date they were determined to
27   have the particular health condition.
28
     ORDER RE SUMMARY JUDGMENT-   523

1   and therefore, closer to the emission sources than Ringold.

2   Because Ringold represents the maximum dose location

3   according to HEDR, Dr. Goble applied his "interindividual

4   variability" analysis only to "individuals residing at Ringold."

5   He could have just as easily calculated 90th, 95th and 99th

6   percentile doses for locations further away than Ringold, or

7   closer to Ringold. Consequently, plaintiffs are bound by Goble's

8   doses as reported in their "Table II: Dr. Goble's Hypothetical

9   Organ Doses for Highly-Exposed Individuals Compared With

10   Defendants' Estimates."

11   The clear implication from Goble's selection of Ringold is

12   that 99th percentile doses in locations further away than Ringold

13   would not be as high as the 99th percentile doses for Ringold.

14   Therefore, the only plaintiffs who can possibly get to trial

15   because of plutonium exposure are: 1) individuals who resided

16   continuously in Ringold from 1944 through 1987, suffer from bone

17   cancer, and were exposed to more than 167 rem; and 2) individuals

18   who resided continuously in Ringold from 1944 through 1987,

19   suffer from lung cancer, are non-smokers, and were exposed at age

20   10 or older to more than 77 rem.[422]

---

[422] A question arises whether Goble has any method for
calculating doses for locations closer than Ringold since Ringold
was the closest location among HEDR's representative locations.
Goble apparently proposes use of a "location adjustment," but
according to defendants, has never applied it to any geographical
location.
        A second question is how long would an individual need to
have resided continuously at Camp Hanford in order to receive a
dose in excess of the applicable doubling dose. The 99th
percentile doses at Ringold require the individual to have
resided continuously there over a 40 plus year period. The court
takes judicial notice that Camp Hanford did not exist for any

ORDER RE SUMMARY JUDGMENT-   524

1   There are a couple of additional things which must be

2 pointed out about Goble's organ doses.   In the tables presented

3 in his November 1997 declaration, Goble says the doses listed

4 apply to **adult** individuals residing at Ringold."   However, Goble

5 agrees with Frazier that "doses to a child from plutonium, unlike

6 the case of iodine, will generally be somewhat smaller, but not

7 appreciably different from the doses to an adult."   (Goble 1997

8 Declaration at p. 4, n. 6).   Consequently, plaintiffs cannot

9 argue that any of the plutonium doses reported by Goble would

10 actually be larger for children.   In fact, they would more likely

11 be smaller because of the undisputed fact that children inhale

12 less air than adults.

13   In his declaration, Goble states that "doses to a particular

14 organ from the river pathway, from plutonium and from iodine (and

15 other fission products) should in principle, be combined."

16 According to Goble, there are a "few organs for which more than

17 one pathway may contribute significantly."   He adds, however,

18 that **"detailed hypothetical combinations would not . . . be**

19 **particularly informative."**   (Goble November 1997 Declaration at

20 p. 10)(Emphasis added).

21   In this case, any reliably calculated non-iodine dose from

22 river exposures can be added to the Ringold individual's

23 inhalation dose for the purpose of determining whether the

24 doubling dose is exceeded for bone cancer or lung cancer.

25 However, for reasons already stated, the court believes any such

26 ─────────────────────────

27 length of time remotely approaching a 40 plus year period.

28 **ORDER RE SUMMARY JUDGMENT-**   **525**

1 doses will be insignificant.

2 In Phase III defendants will be allowed to submit

3 appropriate discovery to plaintiffs' counsel regarding: 1) all

4 plaintiffs with bone cancer or lung cancer who have resided

5 continuously in Ringold from 1944 to 1987; and 2) for lung cancer

6 claimants, those who have been non-smokers (presumably throughout

7 their lives) and were exposed at age 10 or over.

8 If there are any such plaintiffs, then additional discovery

9 can be conducted and determination can be had whether any of

10 those plaintiffs in fact were exposed to doses in excess of the

11 doubling doses for bone cancer and lung cancer[423] based on: 1)

12 the air concentration of plutonium to which the particular

13 individual may have been subject; 2) the breathing rate of the

14 particular individual; and 3) the inhalation dose factor

15 applicable to that particular individual; and 4) reliably

16 calculated river exposures. Of course, only if there is

17 sufficient proof that the individual has been exposed to a dose

18 greater than the applicable doubling dose will his/her bone

19 cancer or lung cancer claim be allowed to proceed to trial.[424]

20 ————————————

21 [423] There is a significant gap between Goble's 95th
percentile and 99th percentile doses for bone and lung cancer.
For bone cancer, the 95th percentile dose is 120 rem and the 99th

22 percentile dose is 258 rem. For lung cancer, the 95th percentile
dose is 41 rem and the 99th percentile dose is 95 rem. For bone

23 cancer, the individual has to prove exposure greater than 167
rem. For lung cancer, he or she has to prove exposure greater

24 than 77 rem.

25 [424] It is not entirely clear whether Goble's method allows
for determination of an **actual** individual's breathing rate,

26 inhalation dose factor, and the air concentration of plutonium to
which he was exposed in a particular location. If that cannot be

27 done, summary judgment may eventually be appropriate on the

28 **ORDER RE SUMMARY JUDGMENT-   526**

(2) Salivary Gland and Breast Cancer (Lactating Female)

For salivary gland cancer[425], Goble reports a 95th percentile I-131 dose for adults of 39 rem and a 95th percentile dose for children of 86 rem.[426] The assumptions underlying these doses are that the adult or the child lived in Ringold in 1945 (the peak iodine emission year) and consumed milk from a backyard cow. The "95th percentile" dose means there is a 5% likelihood an adult or child living in Ringold in 1945 and consuming milk from a backyard cow received such a dose (39 rem or 86 rem). The applicable doubling doses for salivary gland cancer are 33 rem for adults (ages 20 and over); 17 rem for children (ages 10-19); 10 rem for infants (ages 0-9). Thus, for each age category, the 95th percentile dose reported by Goble exceeds the applicable doubling doses.

Here again, defendants contend Goble should be bound by his mean doses. The mean doses found in Table 4 of Goble's declaration are considerably lower than his 95th percentile

_____

remaining bone and lung cancer claims.

[425] Salivary gland is considered a more likely locale for cancer from iodine exposure because it is so closely located to the thyroid which absorbs the greatest amount of iodine. (Goble 1997 Declaration at pp. 6-7). Radford provides risk co-efficients for salivary gland cancer.

[426] These figures come from Table 4 of Goble's November 1997 Declaration at p. 7. Goble lists mean and 95th percentile doses for adults and children who resided in **Richland** in 1945 and were exposed to iodine by drinking milk from a backyard cow. For salivary gland cancer, the 95th percentile dose for adults is 11.2 rem. For children, it is 24.6 rem. Goble states that "[d]ose estimates at Ringold would be approximately 3-4x higher." The Ringold doses found in plaintiffs' Table II (39 rem and 86 rem) reflect a 3.5 upward adjustment (11.2 x 3.5 = 39.2; 24.6 x 3.5 = 86.1).

ORDER RE SUMMARY JUDGMENT-   527

1 doses. For Richland, the adult mean dose is 3.6 rem and the

2 child mean dose is 7.8 rem. Therefore, for Ringold, the adult

3 mean dose would be 12.6 (3.6 x 3.5) and the child mean dose would

4 be 27.3 (7.8 x 3.5). The 12.6 adult mean dose does not exceed

5 the applicable doubling dose of 33 rem for adults. The 27.3 mean

6 dose for children exceeds the applicable doubling doses of 17 rem

7 for ages 10-19 and 10 rem for ages 0-9. The court notes also

8 that Goble's 95th percentile dose for children who resided in

9 Richland in 1945 and received milk from a backyard cow- 24.6

10 rem - exceeds the doubling doses of 17 rem for ages 10-19 and 10

11 rem for ages 0-9.

12   Defendants contend **"most** claimants did not receive their

13 milk from a backyard cow," and **"many** claimants were not exposed

14 during the peak emission. Therefore, defendants assert Goble's

15 doses via the backyard cow pathway are "hypothetical and provide

16 no basis for assessing the organ doses that plaintiffs contend

17 <u>actually</u> resulted from Hanford iodine emissions."

18   However remote it may be that there are actually plaintiffs

19 who lived in Ringold or Richland in 1945 and drank milk from a

20 backyard cow, salivary gland cancer claims will be allowed to

21 proceed into Phase III individual causation discovery. As noted

22 above, additional exposures could have occurred in years beyond

23 1945 and through additional or different pathways. Even by

24 HEDR's calculations, the cumulative iodine releases far exceed

25 the cumulative non-iodine (plutonium) releases.

26   For the same reason, breast cancer claims of adult females,

27 exposed during lactation periods, will be allowed to proceed into

28
ORDER RE SUMMARY JUDGMENT-   528

1   Phase III.[427]

2

3   **D.   I-131 Dose Calculation**

4   In order to determine whether an individual has received a

5   dose of Hanford radiation sufficient to infer that said radiation

6   is "a more likely than not" cause of his/her health condition,

7   there must be some scientifically reliable mechanism for

8   calculating dose.

9   The defendants contend HEDR (Hanford Environmental Dose

10 Reconstruction Project) is adequate for that purpose.

11 Plaintiffs' experts have prepared reports taking issue with the

12 adequacy of HEDR in various respects.   These reports are the

13 subject of motions in limine filed by defendants.

14

15   **1.   Robert Goble**

16   **a.   Summary of Goble's Methodology**

17   In November 1995, Goble prepared a report entitled

18 _____

19   [427] Like the salivary gland, the lactating breast is close
    in proximity to the thyroid gland. (Goble 1997 Declaration at
20 pp. 6-7). Radford provides a risk co-efficient for breast
    cancer.
21   In allowing these salivary gland cancer and breast cancer
    claims to go forward, the court is not relying on the work of Dr.
22 Peters. Dr. Peter's work is too conclusory in asserting a causal
    connection between I-131 exposure and non-thyroid cancers. She
23 also provides no risk co-efficients. Salivary cancer and breast
    cancer (lactating female) claims are allowed to go forward by
24 virtue of granting plaintiffs all favorable inferences from the
    work of Drs. Radford (Iodine Rpt. at p. 28), Roland A. Finston
25 (Plaintiffs' Ex. 31, Appendix 3 re Iodine Claims) and Goble.
    This work includes risk co-efficients. Defendants seek dismissal
26 of all alleged I-131 related non-thyroid cancer claims based on
    lack of expert proof linking I-131 exposure to such cancers. The
27 court finds there is not such an absence of proof.

28
    ORDER RE SUMMARY JUDGMENT-   529

"Estimating Exposures from Releases of Radioactive Iodine at

Hanford and Implications for Assessing the Significance of these

Exposures in Causing Disease." In March 1996, he followed up

with a supplemental report bearing the same title.

The parties agree on what is essentially involved in Goble's

methodology. Goble uses the basic HEDR (Hanford Environmental

Dose Reconstruction) dose model structure, but he engages in what

plaintiffs refer to as a "calibration" of the model. Plaintiffs

define "calibration" as the "act of adjusting a model or its

output to comport with data collected in the field."

Specifically at issue here is Goble's "calibration" of the HEDR

dose model to comport with the 1946 vegetation data, specifically

the Calendar Year 1946 data set.[428]

HEDR used this 1946 vegetation data, as well as other

historical environmental monitoring data[429], in an effort to

"validate" its dose model. Napier, et al., "Validation of HEDR

Models," (1994) (hereinafter, "HEDR Validation Report")

(Defendants' Ex. 122). These are referred to as "validation

exercises" in which computational model estimates are compared

with field and experimental measurements that are independent of

the measurements used to develop the models. (HEDR Validation

---

[428] The Calendar Year 1946 data set includes "Time series at
Richland, 1946," "Time Series at Kennewick/Pasco, 1946," and
"Time series at Benton City, 1946." Napier, et al., "Validation
of HEDR Models," (1994), at p. vii.

[429] The other "validation" sets for the atmospheric
pathway included "Daily Footprint, April 13, 1946," "Green Run
1949," "Purex 1963," "Krypton-85," and "Thyroid 1945-46."
Napier, et al., "Validation of HEDR Models," (1994), at p. vii.

ORDER RE SUMMARY JUDGMENT-   530

1   Report at p. 1.1). HEDR concluded that the monitoring data, as a

2   whole, validated its dose model. However, the HEDR model did not

3   comport as well with the 1946 vegetation data set. For this

4   reason, Goble "calibrated" the HEDR model based on that data set.

5   Defendants hesitate to call Goble's work a "calibration" of

6   the HEDR dose model. They say Goble referred to "calibration" as

7   involving a change in the assumptions or parameters[430] of the

8   model, but that he did not do this. In his 1995 report, Goble

9   stated:

> The **validation exercises** indicate that the
> [HEDR] model results are in rough agreement
> with the environmental data collected for those
> exercises; this is encouraging evidence that
> the basic modeling approach makes some sense.
> **The exercises also indicate a systematic
> underproduction by the model of average quantities
> of radioactive iodine in vegetation. Since the
> most important pathways for human exposures
> (exposures to contaminated milk, vegetables, and
> other foods) involve iodine on vegetation, the
> data indicate that the model is likely to be
> underpredicting doses.** An efficient use of the
> available data would be to **calibrate**, rather
> than simply **validate** the model. Model parameters
> should be adjusted to provide results which show
> no systematic divergence from the data if the goal
> is providing the best estimates of dose consistent
> with available information.

20   (Goble 1995 Rpt. at pp. 26-27)(Emphasis added).

21   _____

22   [430] A parameter is an arbitrary constant each of whose
     values characterize a member of a system. One of HEDR's
23   parameters is the Dry-weight to Wet-weight conversion factor
     which converts wet weights to dry weights. The "technical basis"
24   for this parameter is that the quantity of human food crops
     consumed must be converted from wet mass to dry mass. Snyder, et
25   al., "Parameters Used In the Environmental Pathways and
     Radiological Dose Modules (DESCARTES, CIDER and CRD Codes) of the
26   Hanford Environmental Dose Reconstruction Integrated Codes
     (HEDRIC), (1994), at p. 6.63. This is also referred to as the
27   HEDR Parameter Report (Defendants' Ex. 97).
28
     ORDER RE SUMMARY JUDGMENT-   531

At his deposition, Goble acknowledged he did not go into the equations in the HEDR computer models to correct parameters or equations he believed were deficient. He also acknowledged not knowing the equations or parameters causing the "underprediction" alleged by him. (Goble Dep. at p. 203). Plaintiffs say that although Goble could not "precisely define the bias in each of the parameters that contribute to the underprediction," he has "identified parameters that are likely to be sources of bias." They add that "[b]y correcting systematic biases in **model outputs**, one is in fact correcting the **effects** of model parameter biases." (Plaintiffs' Response Br. at p. 32, n. 39). (Emphasis added).

Correcting the "effects" of parameters is obviously not the same thing as correcting the parameters themselves. The fact Goble did not adjust or correct parameters does not mean that what he actually ended up doing is methodologically unsound. However, it confirms that what he actually ended up doing is multiplying the HEDR dose results by certain factors to account for the difference between the HEDR dose estimates and the Calendar Year 1946 vegetation data set.

The defendants call these "multiplication" factors because they increase the dose estimates. Plaintiffs call them "correction" factors because, according to plaintiffs, they produce "corrected" doses. Although the parties use different adjectives, there is no dispute about the nature of the factors used by Goble: 1) a vegetation multiplication or correction factor; 2) a distance correction factor; 3) a pasture

ORDER RE SUMMARY JUDGMENT-   532

multiplication factor, or what plaintiffs and Goble refer to as a

correction factor for cow feed-to-milk transfer ratios; and 4) a

source term multiplication or correction factor.

Goble's vegetation factor is derived from a "comparison

between the 1946 vegetation measurements (mostly sagebrush) and

HEDR predictions." (Goble 1996 Rpt. at p. 17). Based on the

1946 vegetation data, Goble concludes HEDR underestimates the

amount of iodine on vegetation by a factor of 8.7 for March

through November 1946, and by a factor of 26.3 for December,

January and February of 1946. (Id. at p. 18).

In addition to assuming that HEDR underpredicted vegetation

concentrations in 1946, Goble's vegetation factor is also based

in part on his assertion that the sagebrush measured in 1946 was

wet, while HEDR made its predictions based on dry sagebrush.

This makes for an even larger difference between model

predictions and actual measurements of iodine. To account for

this difference, Goble used a wet/dry ratio of 2.25 ("the needed

adjustment is the ratio of wet weight to dry weight of

sagebrush"). (Id. at p. 17). The wet/dry ratio of 2.25 is

incorporated in Goble's final 8.7 vegetation factor for March

through November 1946 (3.9 x 2.25) and his 26.3 vegetation factor

for December, January and February 1946 (11.7 x 2.25). 3.9 and

11.7 represent HEDR's "underprediction" before application of the

wet/dry ratio.[431] (Goble Dep. at p. 133).

_____

[431] Goble "scaled" up HEDR dose estimates until they
matched the 1946 vegetation data set in order to account for the
underprediction. The 3.9 and 11.7 figures are the result of this
"scaling."

ORDER RE SUMMARY JUDGMENT-   533

1   Goble's vegetation factor applies to all of HEDR's iodine

2 dose **ingestion** pathways, including milk, beef, eggs, fruit,

3 grain, leafy vegetables and other vegetables. It obviously has

4 no application to the external and inhalation pathways.[432]

5 (Defendants' Ex. 40).

6   For the milk and beef pathways only, Goble proposes a

7 pasture multiplication factor, or what plaintiffs and Goble refer

8 to as a correction factor for cow feed-to-milk transfer

9 ratios.[433] According to Goble, "[p]redicted doses from winter

10 milk compared with summer milk for similar releases of

11 radioactivity and concentrations in the air are smaller by a

12 factor of 15-100 . . . [which] does not accord with Hanford

13 measurements of milk concentrations made in the early 1960s . . .

14 ." (Goble 1995 Rpt. at p. 72). Goble's correction factor

15 "represents an adjustment to reflect the Hanford data which

16 indicates that milk in winter, and milk from cows consuming

17 stored feed will have concentrations of radioactive iodine

18 greater than 1/15 the concentrations observed for cows consuming

19 fresh pasture." (Goble 1995 Rpt. at p. 75). Goble agrees that

20 milk and beef from cows consuming fresh pasture will contain

21 greater concentrations of I-131 than milk and beef from cows

22 _____

23   [432] As discussed previously, ingestion is the most
important pathway for I-131 whereas inhalation is the most
24 important pathway for plutonium.

25   [433] This correction factor is related to the vegetation
correction factor because cows ate vegetation (i.e. grass) on
26 which I-131 was concentrated. Goble's pasture correction factor
is based on data derived from sampling of five dairy farms during
27 ten separate months in 1961-62.
28
ORDER RE SUMMARY JUDGMENT-   534

1 consuming stored feed during the winter months, but that cows

2 spent a greater time on fresh pasture in the winter months than

3 assumed by HEDR in its dose estimates. For November through

4 March 1946, Goble concludes a correction factor of three is

5 appropriate, while for April and October, a factor of 1.5 is

6 appropriate.

7   Applying a correction factor of three to the vegetable

8 multiplication factor of 26.2 for December, January and February

9 1946 increases the total multiplication factor to 78.6 (26.2 x 3)

10 for those months. Applying a correction factor of three to the

11 vegetable multiplication factor of 8.7 for March and November

12 1946 increases the total multiplication factor to 26 (8.7 x 3)

13 for those months. Applying a correction factor of 1.5 to the

14 vegetable multiplication factor of 8.7 for April and October 1946

15 increases the total multiplication factor to 13 (8.7 x 1.5) for

16 those months. (Defendants' Ex. 36).[434]

17   The Calendar Year 1946 vegetation data set, from which Goble

18 derives his vegetation correction factor, is the result of

19 vegetation samples "taken from within 30 miles of the Hanford

20 plant." Specifically, it was taken from four locations within

21 that 30 mile radius: N. Richland, S. Richland, Pasco/Kennewick

22 and Benton City. According to Goble, "because [his] correction

23 factors imply greater deposition in the nearby region, it is

24 likely that the plume of radioactivity is somewhat depleted

25 —————————————————

26   [434] Defendants say the overall multiplication factor for
November 1946 is 13, but Goble clearly applies a correction
27 factor of three which produces an overall factor of 26.

28

ORDER RE SUMMARY JUDGMENT-   535

1 further out . . . . .[and therefore,] [a] quantitative measure of

2 this effect is needed to make realistic dose estimates." (Goble

3 1996 Rpt. at pp. 17-18). Goble's "quantitative measure" is a

4 distance correction factor which assumes more iodine was

5 deposited near Hanford and less was deposited at greater

6 distances from Hanford. Goble's vegetation multiplication factor

7 is reduced based on distance from Hanford. The results can be

8 seen at p. 21 of Goble's 1996 Report and at Defendants' Ex. 38.

9 For example, while Goble increases iodine doses by a vegetable

10 multiplication factor of 8.7 during March through November for

11 locations within 25 miles of the Hanford plant, this factor is

12 reduced to 1.26 for a location 205 miles from the Hanford

13 plant.[435]

14   Goble's "calibrations" are based only on the vegetation data

15 for 1946. He extrapolates his "calibrations" to subsequent years

16 by comparing how HEDR's 1946 iodine release estimates compare to

17 release estimates for other years. Goble chose to use the iodine

18 source term estimate of plaintiffs' experts, Drs. Franz and

19 Brigitte Herrmann, for this purpose. The Herrmanns' release

20 estimate covers the period 1948-60.[436] Defendants' Ex. 39 shows

21

22   [435] Goble's pasture multiplication factor is not included
    in defendants' chart (Ex. 38) and this is perhaps because the
23 assumptions underlying that factor (cows pastured longer between
    October and April) are not true the further one gets away from
24 Hanford. The climate is milder in Richland than it is in Spokane
    or points further north.

25

      [436] For the years 1944-47, Goble uses the HEDR source
26 term release estimates. The component of HEDRIC (Hanford
    Environmental Dose Reconstruction Integrated Codes) pertaining to
27 radionuclide release rates is "STRM."

28
ORDER RE SUMMARY JUDGMENT-   536

the source term multiplication factor for the months of 1951

based on the Herrmanns' monthly estimates for that year. For

January 1951, the source term multiplication factor is 17.3 which

is derived by dividing the Herrmanns' release estimate by the

HEDR release estimate for that month. Thus, the dose estimate

from application of Goble's other factors (vegetation

multiplication factor, pasture multiplication factor, and

distance correction factor) are increased by a factor of 17.3 for

January 1951.

All of the factors are considered together to arrive at an

overall multiplication or correction factor which is then applied

to HEDR's monthly doses. The result is an increase in those

doses.

b. Reliability

Defendants contend Goble falsely assumes the discrepancy

between the Calendar Year 1946 vegetation data set and the HEDR

model predictions is the result of a problem with the model

itself and that none of the discrepancy is due to problems with

the 1946 data. They say that vegetation data collected in

subsequent years using improved measurement techniques confirms

the discrepancy is not due to the model. Defendants contend

Goble's failure to consider the limitations of the 1946 data and

his failure to consider subsequent vegetation monitoring data

makes his methodology unscientific and unreliable.

//

//

ORDER RE SUMMARY JUDGMENT-   537

(1)   Limitations of the 1946 Vegetation Data

The initial procedure used to measure beta radioactivity on vegetation[437] involved preparation of a one-gram pellet of the vegetation sample, placing the pellet on a Geiger-Mueller detector system, and counting the rate of beta particle emissions from the sample (counts per minute per gram or cpm/g). A conversion factor was used to convert the counts of beta emissions rate to number of beta disintegrations per minute per gram of vegetation (dpm/g). This procedure was used from the summer of 1945 until December 1948 and involved the collection of sagebrush samples from the Hanford environs. Gilbert et al., "Uncertainty and Sensitivity Analysis of Historical Vegetation Iodine-131 Measurements in 1945-1947," (1994), at p. 1.1 (hereinafter, "Gilbert 1994").

The initial procedure was a "gross beta" method which could not distinguish between I-131 and other beta-emitting radionuclides which might also be on the sample pellet. Gilbert 1994 reports that until 1948, it was assumed all activity measured on vegetation was from I-131 and "[i]ndeed, the fraction of total radioactivity that was due to iodine-131 was probably very close to 1 in 1945 . . . ." Later on, however, other radionuclides made up a larger fraction of the activity and as a result "the iodine-131 activities . . . that were reported from 1945 to 1948 were **biased** to varying degrees." (Gilbert 1994 at pp. 1.2 and 1.3)(Emphasis added).

---

[437] I-131 emits beta radiation. Pu-239 emits alpha radiation.

ORDER RE SUMMARY JUDGMENT-   538

1   The HEDR Validation Report had this to say about the

2   Calendar Year 1946 data set:

3   With the exception of January, the estimated
         measured time sequences at each location are
4   all within an order of magnitude throughout the
         year of 1946. They are closest during the
5   important summer grazing season, with all monthly
         measurements being within factors of 3 of
6   estimated medians (with one exception in south
         Richland and two in Benton City). **Each time
7   sequence shows the largest deviation between
         estimates and measurements during the winter
8   months, with January and December approaching
         factors of 10 underestimates at all locations.**
9   For those cases of greatest underestimate, the
         monthly means of the measurements are all
10   within factors of 3 or less of the extremes of
         the estimates.

11   (HEDR Validation Report at p. 3.3)(Emphasis added).

12   The Validation Report points out that due to use of the

13   Geiger-Mueller detector system, the "uncertainty in the

14   conversion of [the 1946] count data to concentration could be a

15   factor of up to 4 for this period."

16   Defendants cite passages from Goble's deposition as evidence

17   that he "completely ignore[d] the important limitations of the

18   1946 measurements." Goble was asked whether he analyzed HEDR's

19   explanation of the uncertainty involved in the 1946 vegetation

20   data. His response was that he did not recall "very much

21   discussion of that" in his own reports. He testified he did not

22   recall discussing in his report the quantitative uncertainty

23   levels provided in Gilbert 1994 (PNWD-1978). He acknowledged

24   that his reports did not quantitatively compare the uncertainties

25   involved in the vegetation data compared to the uncertainties in

26   the HEDR model. He did not incorporate the uncertainty in the

27
28   **ORDER RE SUMMARY JUDGMENT-   539**

1   1946 vegetation data into his calibration factors.   He did not

2   incorporate Gilbert's probability distribution for the

3   uncertainty of the vegetation data into his uncertainty analysis

4   for his calibration procedure.   (Goble Dep. at pp. 99-102).

5   Goble stated his "overall uncertainty analysis include[d] an

6   assessment of the uncertainty in [his] **calibration procedure**,"

7   but that he "did not make a quantitative analysis" for the

8   uncertainty in the vegetation data.   He asserted the uncertainty

9   of the vegetation data was "within the overall uncertainties of

10 the whole approach."   (<u>Id</u>. at p. 102)(Emphasis added).

11   Defendants say Goble makes no attempt to analyze how much of

12 the discrepancy between the 1946 vegetation data and the HEDR

13 model predictions is due to the data rather than the model.

14 Rather, say defendants, he simply assumes the entire "winter

15 discrepancy" arose from the model.[438]   Defendants contend this

16 is contrary to Goble's own acknowledgement that in comparing an

17 environmental dose reconstruction model to environmental

18 monitoring data, a relevant question is the quality of the

19 monitoring data (Goble Dep. at p. 64); and his statement that it

20 was "desirable" to "explain coherently how much of the

21 uncertainty and limitations is attached to specific data sets . .

22 . ."   (<u>Id</u>. at p. 79).

23   Plaintiffs argue that Goble "clearly" considered the

24 limitations and the uncertainty of the 1946 vegetation data.

25 _____

26   [438]   "Winter discrepancy" refers to HEDR's finding that the
    highest deviations between model predictions and the Calendar
    Year 1946 measurements occurred during the winter months,
27 particularly January and December of 1946.

28 **ORDER RE SUMMARY JUDGMENT-   540**

1 They cite Goble's deposition testimony wherein he states that he

2 "used the key conclusions of [Gilbert 1994] which were that the

3 uncertainties [of the vegetation data] were modest in comparison

4 with the uncertainties in the model . . . ." (Goble Dep. at p.

5 99). What Goble was alluding to is the following passage from

6 Gilbert 1994:

> 7 [T]here may be no need to reduce the
>   uncertainty of the historical vegetation
>   iodine-131 concentrations. The primary
> 8 use of the historical vegetation iodine-131
>   concentrations is to help validate the HEDR
> 9 Project source-term, air-transport, and
>   environmental accumulation models being used
> 10 to compute vegetation iodine-131 concentrations
>   in the study area as an intermediate step in
> 11 computing doses to individuals from exposure
>   to iodine-131 via the air pathway. **The un-**
> 12 **certainties in the predicted vegetation iodine-**
>   **131 concentrations obtained on the basis of**
> 13 **these models are likely to be very large because**
>   **of the large uncertainties in some model parameter**
> 14 **values. The uncertainties in the model predicted**
>   **iodine-131 concentrations are likely to be so**
> 15 **large that reducing the uncertainties in converting**
>   **measured historical cpm/g radiation measurements to**
> 16 **iodine-131 concentrations will not perceptibly**
>   **affect the conclusions of the validation effort.**

18 (Gilbert 1994 at vii)(Emphasis added).

19   Plaintiffs also cite to another HEDR document, Mart, et al.,

20 "Conversion and Correction Factors for Historical Measurements of

21 Iodine-131 in Hanford-Area Vegetation, 1945-1947," (1993). This

22 report provides conversion factors for converting original

23 counting data to numerical values representing the best

24 approximation of the actual amounts of I-131 deposited onto

25 vegetation at the Hanford site and surrounding areas. Id. at p.

26 vi. According to the report:

27   While these results are necessarily estimates,

28 **ORDER RE SUMMARY JUDGMENT-   541**

1   they provide a basis for estimating the overall
     uncertainty in the available vegetation data
2   for the 1945-1947 period. **Because the uncertainty
     in the total conversion factors . . . relative to**
3   **the uncertainty in the models and model parameters
     being developed to predict radiation doses is small,**
4   **further refinement of these vegetation estimates is
     not deemed appropriate nor planned for the balance of**
5   **this project.**

6   (<u>Id</u>. at p. 7.3).

7   The thrust of plaintiffs' arguments appears to be that

8   because HEDR was content to live with some uncertainties

9   surrounding the 1946 vegetation data, it is likewise okay for Dr.

10   Goble to accept those uncertainties for the purpose of his

11   analysis. Plaintiffs say Goble merely relied on "quality

12   assurance work done by HEDR" and that the 1946 vegetation data is

13   "quality assured data" due to HEDR's efforts.

14   Plaintiffs' arguments ignore the fact that whereas HEDR

15   considered vegetation data other than the 1946 data, including

16   data from subsequent years using improved tests for measuring I-

17   131, Goble considered **only** the 1946 data in his tweaking of dose

18   estimates.[439] The uncertainties of the 1946 data have much more

19   significant consequences for Goble's analysis because it is based

20   _____

21   [439] The plaintiffs cannot deny the Calendar Year 1946 data
     set is the cornerstone of Goble's dose estimation analysis.
22   Asked at his deposition whether his method essentially
     substituted the 1946 vegetation data for HEDR's vegetation
23   predictions, Goble stated:

24   . . . what my method does is uses the HEDR
          model to make predictions where the model
25   has been modified in **one** aspect of it to
          make it **conform to the average of the**
26   **[1946] vegetation measurements.**

27   (Goble Dep. at p. 114).
28
     ORDER RE SUMMARY JUDGMENT-   542

1 solely on that data.

2   HEDR emphasized the fact that it relied on a **series** of

3 validation exercises:

> Because not enough data are available, no
> **individual validation exercise adequately
> verifies the accuracy of the HEDR computer
> models.** It is only through the compilation
> **of a sufficient number of component validations
> that the reliability of the HEDR computer models
> is demonstrated.** The results of <u>all</u> of the
> validation tests that have been performed combine
> to provide a reasonable validation set for the
> needs of the project. Sufficient coverage of
> the spatial, temporal, and pathway variables
> is achieved and demonstrates a high level of
> **confidence in the adequacy of the HEDR approach
> and implementation.** On the basis of the tests
> performed and the results obtained, the staff
> of the HEDR Project conclude that the models in
> the HEDR toolbox are fully functional and accurate.
> These models meet the HEDR Project objectives in
> that they provide sound, supportable estimates
> of individual radiation doses resulting from
> historical releases of radionuclides from the
> Hanford Site. As a result of this validation
> exercise, no revisions to any of the models are
> recommended before estimation of representative
> individual doses.

17 (HEDR Validation Report at p. viii).[440]

18   The fact HEDR considered the 1946 vegetation data set along

19 with other data in an effort to validate its doses over a 50 year

20 period does not make it per se scientifically appropriate for

21 Goble to have used **only** that data set in his calibration

22 analysis. Nonetheless, that is precisely the rationale Goble

23 ──────────────────

24   [440] The Validation Report states that "contemporaneous
data" did not address all the necessary pathways, either
25 geographical or temporal, to provide a complete validation and
therefore, the data sets selected were chosen to provide the best
26 examples of the coverage of the HEDR Project domain in space, in
time, and for as many pathways as possible. (HEDR Validation
27 Report at p. v). (Emphasis added).

28 **ORDER RE SUMMARY JUDGMENT-   543**

1 uses.   At his deposition, Goble acknowledged there was a "certain

2 danger of selectivity when you . . . pick out one piece of or one

3 small data set . . . ." However, Goble felt this was not a

4 problem because he was "relying on someone's else's selection of

5 the data . . . ." (Goble Dep. at p. 96). Plaintiffs' counsel

6 employ the same argument in their brief, contending Goble

7 properly relied on "quality assured data" from HEDR. The point

8 is, however, that Goble did something different than HEDR. Goble

9 used **only** the 1946 vegetation data set for modifying HEDR doses

10 over an approximate 25 year period (1944-60).

11   The plaintiffs contend defendants have no expert or

12 documentary support for their assertion that the discrepancy

13 between HEDR model predictions and the 1946 vegetation data is

14 due to limitations of early measurement methods. Plaintiffs say

15 this is pure speculation on the part of defendants. Plaintiffs

16 note the underprediction for the winter months of 1946 is

17 significantly higher than for the summer months. They assert

18 this fact indicates the underprediction is due to the HEDR model

19 itself. According to plaintiffs:

20   If the underprediction were due to 'serious
            limitations' of the early Hanford sampling
21   techniques and instrumentation, then these
            limitations would affect all months of the year
22   1946 equally, and there should be no seasonal
            pattern in HEDR underpredictions. The fact that
23   there is a seasonal pattern in underprediction
            argues that it is the model which is biased
24   and not the underlying data.

25 (Plaintiffs' Response Br. at p. 44).

26   Plaintiffs cite no expert or documentary support for this

27 assertion. There is no indication that Goble advanced this as a

28 **ORDER RE SUMMARY JUDGMENT-   544**

reason to disregard the limitations of the early measurement techniques. Insofar as plaintiffs' contention that defendants offer no documentary support that the discrepancy is due to limitations of the early measurement techniques, that is not the case.

As noted, the HEDR Validation Report states the uncertainty of the conversion of the count data from the Geiger-Muller detector to concentration could be a factor of up to 4 for calendar year 1946. It adds that "[o]nly the deterministic 'best estimate' of Mart et al. (1993) . . . has been used in these analyses" and that "[i]ncorporation of this uncertainty in the analyses would indicate a greater overlap than is apparent in the figures." (HEDR Validation Report at p. 3.14).

With regard to the April 13, 1946 data set (known as the "footprint data")[441], the Validation Report states that the "comparison of the estimates to the measurements is hindered by the poor resolution of the detection equipment used in 1946." Id. at p. 4.6.

With regard to the December 1949 Green Run data set, the Validation Report observes that "techniques for radionuclide detection in environmental samples has improved over those available in 1946" and that the new "multi-step chemical extraction process" (aka "wet chemistry") provided a "much better counting geometry and reduced the uncertainty of absorption of beta emissions within the sample." (Id. at p. 5.3). Goble

---

[441] To be distinguished from the Calendar Year 1946 data set.

ORDER RE SUMMARY JUDGMENT-   **545**

1 acknowledged at his deposition that an advantage of the chemical

2 extraction process was that it avoided confusing I-131 with other

3 beta-emitting radionuclides.   (Goble Dep. at p. 107).   He also

4 acknowledged an advantage because of the "simpler geometry"

5 involved with a "two-dimensional sample" rather than a "three-

6 dimensional sample."   (Id. at p. 108).

7   Finally, the "Conclusions" section of the HEDR Validation

8 Report includes the following statement:

9   The comparisons of estimates to measurements
            for the four 1946 time series for north Richland,
10   south Richland, Kennewick/Pasco, and Benton City
            . . . show order-of-magnitude agreement.   The
11   comparisons for 1946 correlate to within factors
            of 3 during the grazing season months.   Comparisons
12   during winter months are not as close.   **The historical
            measurements themselves, however, are of variable
13   quality.   Comparisons made for later times . . . are
            better, in part possibly because monitoring methods
14   for the later periods were improved.**   The largest
            underestimates are at depositions greater than 300
15   nCi/kg, which occur during the winter months.
            However, the DESCARTES[442] results tend to overestimate
16   the deposition resulting from the Green Run (also
            a December/winter release resulting in comparable
17   deposition).   **Differences in the data sets include the
            way that the contamination was measured at the time and
18   the regional weather during the release.   This result
            argues against a systematic error.**[443]

19

20 (HEDR Validation Report at p. 15.1)(Emphasis added).[444]

21   [442] DESCARTES is the component of HEDRIC which measures
    radionuclide concentration in vegetation and in animal products.

22
         [443] Goble, of course, contends there is a "systematic"
23 error.

24   [444] According to Denham, et al., "Conversion and Correction
    Factors for Historical Measurements of Iodine-131 in Hanford-Area
25 Vegetation 1948-51," (1993), p. vii:

26   Because of the improved method of analyzing
            iodine-131 that was developed and initially
27   implemented in December 1948, the uncertainties
28
    ORDER RE SUMMARY JUDGMENT-   546

1   Plaintiffs note the HEDR Validation Report specifically

2 indicates in "Table S.1 Degree of Validation Obtained for the

3 Atmospheric Release Pathway Models" that for the April 13, 1946

4 Data Set ("Dispersion/Deposition Footprint"), the "[q]uality of

5 historical measurements impacts comparison." (HEDR Validation

6 Report at p. vii). The table does not indicate the same for the

7 Calendar Year 1946 data set. On the other hand, there is no

8 denying that the same measurement technique was used for the

9 Calendar Year 1946 data set and for the April 13, 1946 data set.

10   Plaintiffs go on to argue as follows:

11   The only 'assumption' Dr. Goble made, is
            that HEDR correctly analyzed the technical
12   issues, with regard to the 1946 vegetation
            monitoring techniques, and produced a
13   reasonably accurate 'best estimate' of the
            raw count data to concentration conversion
14   factor. Dr. Goble relied on HEDR's analysis
            of the 1946 vegetation data, which provided
15   a 'best estimate' (statistically unbiased)
            of the historic vegetation contamination
16   levels. Thus, the discrepancy between HEDR
            predictions and the HEDR statistically un-
17   biased 'best estimate' of the vegetation
            contamination, by definition, is due to
18   biases in the model.

19 (Plaintiffs' Response Br. at p. 46).

20   There is no indication from plaintiffs that Goble made such

21 _____

22   reported . . . for the 1945-1947 conversion
            and correction factors **should decrease for the
23   1948-1951 period. Therefore, the degree of uncertainty
            of the reconstructed iodine-131 levels that would
24   be obtained using the conversion and correction
            factors reported here will not specifically be
25   addressed because the improved method of analysis
            should <u>drastically</u> reduce the uncertainty associated
26   with individual analyses.**

27 (Emphasis added).
28
ORDER RE SUMMARY JUDGMENT-   547

1 an assertion in any of his expert reports or at his deposition.

2 There is no affidavit or declaration from Goble asserting such.

3 This is the argument of plaintiffs' counsel.[445]

---

[445] The court believes there may be a very good reason why
Table S.1 of the HEDR Validation Report says that for the April
13, 1946 data set the "quality of historical measurements impacts
comparison," but does not say the same for the Calendar Year 1946
data set. As noted, HEDR did not incorporate the uncertainty in
the conversion of count data to concentration in its comparison
of the Calendar Year 1946 data set with the HEDR model
predictions. The HEDR Validation Report pointed out that the
uncertainty factor could have been up to 4 for this period
(1946).
     In its comparison of the HEDR model predictions with the
Calendar Year 1946 data set, HEDR used only the "deterministic
'best estimate' of Mart, et al. (1993)." Mart 1993 described the
reconstructed conversion and correction factors for historical
measurements of I-131 in Hanford area vegetation collected
between October 1945 and December 1947. These factors could be
used to derive "best estimates of true iodine-131 activities in
the historical vegetation samples." (Mart 1993 at pp. v and vi).
These are the "best estimates" referred to by plaintiffs' counsel
which they say Goble is entitled to take at face value.
     Mart 1993 states as follows:

          The results of this report [Mart 1993] must be
          viewed in light of the limitations in the scope
          of this report. While the results are necessarily
          approximations, they do not provide a basis for
          estimating the overall uncertainty in the
          available vegetation data for 1945-1947. Although
          additional research would reduce the uncertainty
          in the conclusions, the uncertainty in the
          conversion factors (Gilbert et al. 1992) relative
          to the uncertainty in the models and model parameters
          being developed is small. Therefore, further refine-
          ment of these vegetation estimates is not necessary.

(Mart 1993 at p. vi). It may be for this reason the HEDR
Validation Report used only the "deterministic 'best estimate'"
of Mart 1993 and did not incorporate the uncertainty in the
conversion factors reported by Gilbert et al. 1992.
     The HEDR Validation Report concluded the incorporation of
that uncertainty would indicate "a greater overlap than is
apparent in the figures" (the "figures" being the HEDR model
predictions as compared to the historical measurements found in
the Calendar Year 1946 data set). It reasonably appears what is
meant by "a greater overlap" is that if the uncertainty (the
uncertainty arising from limitations in the measurement

ORDER RE SUMMARY JUDGMENT-   548

1

2   **(2)   Subsequent Vegetation Data**

3   Defendants contend vegetation data collected in years

4   subsequent to 1946, after the development of improved measurement

5   techniques, refutes the claim that the discrepancy between HEDR

6   model predictions and the 1946 vegetation data set is due to the

7   model, rather than limitations of the measurement techniques

8   available in 1946.

9   One of the "subsequent" vegetation data sets used by HEDR to

10 validate its model was the Green Run of 1949. The Green Run

11 involved an intentional release of 7,000 curies of I-131 to the

12 atmosphere over a "brief period in December 1949." Extensive

13 environmental monitoring took place in the weeks following the

14 release.   (HEDR Validation Report at p. 5.1).

15   According to the HEDR Validation Report:

16   By 1949, techniques for radionuclide detection in
             environmental samples had improved over those
17   available in 1946. Concentration measurements
             of iodine-131 in vegetation were made with a
18   multi-step chemical extraction process, in which
             iodine-131 was removed from the sample and the
19   resulting solution counted.[446] This provided
             much better counting geometry and reduced the
20   uncertainty of the absorption of beta emissions
             within the sample.

21

22 _____

    techniques available in 1946) had been incorporated into
23 comparison between the figures, it would have **reduced** the
    discrepancy between HEDR model predictions and the historical
24 measurements for Calendar Year 1946. There would have been
    "greater overlap" between model predictions and historical
25 measurements meaning limitations in the measuring techniques
    available in 1946 in fact affected the comparison.

26

27   [446] This is otherwise known as the "wet chemistry" approach.

28

ORDER RE SUMMARY JUDGMENT-   549

1 (Validation Report at p. 5.3).

2   In his 1995 report, Goble had this to say about the 1949

3 Green Run data:

> The December 1949 Green Run experiment is
> relatively well documented (Jenne 1950). Un-
> fortunately little attempt was made during the
> experiment to determine its environmental im-
> pacts; the emphasis instead was in assessing
> radiation detection capabilities. As with the
> April 13 [1946] data, there is a substantial
> discrepancy between the model predicted location
> of **peak** concentrations and the observations.
> **Especially since this was a single release, it
> is difficult to interpret this as indicating
> an inadequacy of the model to predict transport
> on the** _**average**_. The two tests, however, do provide
> convincing evidence that the model does not track
> actual transport closely, and one should not
> expect to be able to base predictions on a very
> tight correlation of releases with meteorological
> conditions. **The comparison of** _**average**_ **concentrations
> shows that the model only under predicts in this case
> by a factor of 1.3.** As noted by the authors, (Napier
> 1994) p. 5.12[447], the agreement with the model is
> best for the early period. Indeed the model tends
> to significantly underestimate concentrations later
> on, consistent with longer retention times for
> contamination in the vegetation. **The approximate
> agreement at the beginning of December, contrasts
> with the large discrepancies found in winter in 1946.**
> One difference is that the retention of iodine plays
> a different role in the two cases; a second difference
> is that the weather conditions were very carefully
> selected for the [Green Run] experiment (indeed the
> experiment was delayed for a week in November because
> of inclement weather involving fog . . .).

21 (Goble 1995 Report at pp. 57-58)(Emphasis added).

22   In this passage of his report, Goble says nothing about the

23 change in measurement techniques from 1946 (Geiger-Mueller

24 detector) to 1949 ("wet chemistry"). Indeed, plaintiffs do not

25 cite a single sentence from any of Goble's reports referring to

---

27   [447] The HEDR Validation Report.

28

ORDER RE SUMMARY JUDGMENT-   550

1 the change in measurement techniques. At his deposition, Goble

2 acknowledged that neither of his reports discussed the methods

3 for counting vegetation data. (Goble Dep. at pp. 103-04).

4   Defendants assert Goble's statement is an acknowledgement by

5 him that his 11.7 underprediction for the winter months in

6 1946[448] had been reduced to a factor of 1.3 after the

7 introduction of the improved "wet chemistry" measurement

8 technique in 1948. According to defendants, despite Goble's

9 acknowledgement of the "superior and more reliable quality of the

10 1949 Green Run data," he disregards it because it would show that

11 his calibrations overpredict the December 1949 vegetation

12 concentrations by a factor of 7 using the Herrmanns' I-131 source

13 term estimate, and by a factor of 12 using Klementiev's I-131

14 source term estimate. Defendants contend Goble's disregard of

15 primary data contrary to his opinion violates a well-established

16 rule pertaining to scientific reliability.

17   Defendants contend Goble also violated this rule by

18 disregarding admittedly superior vegetation data for 1948-51, and

19 by failing to compare his dose method to any of the vegetation

20 data collected at Hanford from 1951 through 1983 using superior

21 measurement techniques.

22   According to plaintiffs, although Goble considered the 1949

23 Green Run Data, he did not incorporate it into his "calibration"

24 because the "Green Run was not an accurate test of model

25

---

26   [448] 11.7 is the underprediction before application of the
wet/dry ratio which increases the vegetation correction factor to
27 26.3 (11.7 x 2.25) for the winter months.

28
ORDER RE SUMMARY JUDGMENT-   551

1   performance over average or long-term periods." In other words,

2   plaintiffs assert Goble's point is "that for the Green Run only,

3   not routine operational periods, the Green Run data may be

4   preferable for dose estimates." Plaintiffs argue that Goble

5   clearly shows the Green Run data should not be incorporated

6   "quantitatively" into any assessment of long-term, average doses

7   to individuals, but that the Green Run data "qualitatively"

8   indicates the HEDR model will underpredict vegetation

9   contamination over average conditions for long periods, because

10   HEDR's assumption on the iodine retention time on vegetation is

11   biased low. In support of these arguments, they cite the passage

12   of Goble's report quoted above (Goble 1995 Report at pp. 57-58).

13   This is an interpretation of Goble's statement offered by

14   plaintiffs' counsel. Goble does not offer such an interpretation

15   in any type of declaration in the record before this court.

16   There is nothing explicit in Goble's 1995 report (pp. 57-58) that

17   the Green Run data should be excluded for the purpose of

18   estimating doses for "routine operational periods." While Goble

19   says the Green Run data shows a "substantial discrepancy between

20   the model predicted location of **peak** concentrations and the

21   observations" (i.e. the measurements), he qualifies his remark by

22   adding that because the Green Run "was a single release, it is

23   difficult to interpret this **as indicating an <u>inadequacy</u> of the**

24   **model to predict transport on the <u>average</u>**." (Emphasis added).

25   In other words, the existence of substantial discrepancies

26   between HEDR model predictions of peak I-131 concentrations and

27   historical measurements for the single release Green Run does **not**

28   **ORDER RE SUMMARY JUDGMENT-**   **552**

1 necessarily mean the HEDR model will underpredict concentrations

2 on "average" (i.e. over the long run).

3   Goble admits the Green Run data shows agreement between

4 historical measurements and model predictions for the "beginning

5 of December," which "contrasts with the large discrepancies found

6 in winter in 1946." Goble attributes the difference to "the

7 retention of iodine" and the fact weather conditions were

8 carefully selected for the Green Run. With regard to the latter,

9 plaintiffs say that because the Green Run avoided fog, post-Green

10 Run vegetation data is not representative of vegetation

11 contamination levels on "average" during the winter. On the

12 other hand, they say the Calendar Year 1946 data set includes

13 vegetation measurements for iodine that would have been deposited

14 during foggy conditions in the winter months. The HEDR

15 Validation Report indicates damp weather, including fog, mist and

16 hoarfrost, may increase the deposition or retention of I-131 on

17 vegetation. (HEDR Validation Report at p. 3.3).

18   Essentially, plaintiffs' contention is the large discrepancy

19 between model predictions and the historical measurements during

20 the winter months of 1946 is due to the possibility that fog

21 increased deposition on the vegetation at that time, and the

22 iodine stayed on the vegetation longer. Both of these were

23 factors apparently not considered by HEDR. The HEDR Validation

24 Report indicates "several potential deposition mechanisms were

25

26

27

28

ORDER RE SUMMARY JUDGMENT-   **553**

1 not included in RATCHET[449] and DESCARTES," one of them being

2 fog.   (HEDR Validation Report at p. 3.3).

3   At his deposition, Goble testified that in calculating his

4 adjustment for distance, he assumed an increase in retention time

5 for iodine based in part on the Green Run Data and in part on the

6 PUREX event.   (Goble Dep. at pp. 93-94).   According to Goble's

7 1996 Report, his approach included "a component reflecting

8 greater storage of iodine on the vegetation than is assumed in

9 most runs of the HEDR model."   Goble indicated there was good

10 evidence this component should be increased by a factor between

11 1.5 and 2.0.   An increase by a factor of 2.0 was the maximum

12 possible, according to Goble, because the radioactive half-life

13 of I-131 is 8 days and the effective life that appears in the

14 HEDR runs is about 4 days.   (Goble 1996 Report at p. 19 and n.

15 10).

16   In sum, plaintiffs suggest that because the HEDR model did

17 not adequately take these factors (iodine retention and fog) into

18 account, that is why the model so significantly underpredicts

19 doses for the winter of 1946 in comparison to what is shown in

20 the 1946 historical measurements.   This is as opposed to

21 inadequacies in the 1946 measurement techniques potentially being

22 responsible for the discrepancy- i.e. the historical measurements

23 being biased high because all the beta emitting radionuclides, as

24 opposed to just I-131, were counted.

_____

26 [449]   RATCHET is the component of HEDRIC which measures
radionuclide concentrations in the air and radionuclide
27 concentration rates.

28
ORDER RE SUMMARY JUDGMENT-   554

1   For the Green Run specifically, plaintiffs suggest the

2   significantly lessened discrepancy between the HEDR model

3   predictions and the historical measurements for that time period

4   (December 1949) is due to the avoidance of fog, as opposed to the

5   existence of the "wet chemistry" measurement technique.

6   Furthermore, plaintiffs say retention time is not as significant

7   a factor with regard to December 1949 historical measurements

8   because of the narrow time period involved which also lessens the

9   discrepancy of model predictions and historical measurements for

10   that narrow time period (December 1949). However, plaintiffs say

11   that using the Green Run data over "average conditions for long-

12   periods" will cause an underprediction of vegetation

13   concentrations because of HEDR's underestimation of retention

14   time.

15   The court is in no position to say that fog or retention

16   time can be ruled out as factors. Although defendants may not

17   agree with Goble about his retention time correction, they do not

18   tender any arguments against it. Plaintiffs assert with

19   confidence that the Calendar Year 1946 data set would certainly

20   have included foggy conditions, but there is no indication about

21   the extent to which it was foggy during that year and therefore,

22   the extent to which fog would have affected deposition.

23   Retention time and the existence of foggy conditions may

24   well be legitimate bases for explaining the discrepancy between

25   HEDR model predictions and historical measurements for 1946.

26   However, that does not necessarily excuse considering the

27   improvement in measurement techniques between 1946 (Geiger-

28   **ORDER RE SUMMARY JUDGMENT-   555**

1 Mueller) and 1949 (wet chemistry) as a legitimate basis for some

2 or all of the discrepancy. As noted, Goble readily acknowledged

3 this improvement in the measurement techniques.

4   Goble's distance correction factor, "a quantitative

5 measure," assumes an increase in retention time. (Goble Dep. at

6 pp. 93-94; Goble 1996 Report at p. 19). Increased retention time

7 is a "component" of Goble's distance correction factor. Two

8 other components are: 1) a greater deposition rate of

9 radioactivity than is assumed on average in the runs of the HEDR

10 model; and 2) a component reflecting greater interception of the

11 iodine by vegetation relative to the overall deposition rate.

12 For his "winter correction, which may well result in part from

13 special conditions such as fog," Goble **increased** these

14 components. (Goble 1996 Report at p. 19). In other words, it

15 appears Goble effectively quantified increased retention time and

16 the possible existence of fog, the effect of which is to increase

17 dose estimates. At the same time, however, he did not quantify

18 the uncertainty about the accuracy of the 1946 measurement

19 technique, the effect of which would possibly have **decreased** his

20 dose estimates.

21   When the plaintiffs finally get around to discussing the

22 "wet chemistry" technique used in the Green Run historical

23 measurements, they argue defendants have failed to advise that

24 HEDR developed conversion and correction factors to account for

25 biases in vegetation sampling for the "wet chemistry" method,

26 just as it did for the gross beta (Geiger-Mueller) method used in

27 1946. Denham, et al., "Conversion and Correction Factors for

28

ORDER RE SUMMARY JUDGMENT-   556

1 Historical Measurements of Iodine-131 in Hanford Area Vegetation

2 1948-1951," (1993)(PNWD-2176 HEDR).[450]

3   The fact conversion and correction factors were necessary to

4 reconstruct true iodine-131 activity levels in vegetation for

5 1948-51 does not necessarily excuse Goble from considering the

6 uncertainty arising from the measurement technique (Geiger-

7 Muller) used in conjunction with vegetation sampling in 1946.

8 This is especially so when the 1946 vegetation data is the

9 cornerstone of Goble's dose estimation method. Plaintiffs do not

10 cite to anything in Goble's reports, nor any other expert

11 reports, as support for their argument that biases in the "wet

12 chemistry" method would excuse consideration of biases in the

13 1946 Geiger-Muller method.

14   Plaintiffs seem to suggest that the biases involved in the

15 two methods cancel each other out, but that is clearly not the

16 case. HEDR recognized the greater reliability of the "wet

17 chemistry" method and so did Goble. Indeed, Denham I 1993

18 concluded that "[t]he overall detection efficiency, as indicated

19 by the reconstructed measurement factor (M), **increased**

20 **approximately an order of magnitude between the 1945-1947 pellet**

21 **analysis technique and the 1948-1951 precipitate analysis."**

22 (Denham I 1993 at p. vi)(Emphasis added).

23   Attached to plaintiffs' response brief is an "Appendix A" in

24 which there is a discussion of the "gamma spectrometry"

25 measurement method which eventually succeeded the "wet chemistry"

26 ───────────────

27   [450] Hereinafter, "Denham I 1993."

28 **ORDER RE SUMMARY JUDGMENT-   557**

1 method in the late 1950s. Plaintiffs cite various sources as

2 indicating that use of the gamma spectrometry method showed I-131

3 contamination levels discrepant with the levels shown by use of

4 the "wet chemistry" method. According to plaintiffs, Hanford

5 records indicate "wet chemistry" results were retroactively

6 increased by a factor of three when the discrepancy was

7 discovered and "all historically reported 'wet chemistry'

8 vegetation contamination results **may** have to be increased by a

9 factor of three to produce reliable historical vegetation

10 contamination levels."

11   Here again, any limitations of the "wet chemistry" method do

12 not necessarily make the limitations of the Geiger-Muller pellet

13 analysis disappear. The fact is that Goble did not consider or

14 quantify the limitations of the Geiger-Muller pellet analysis in

15 finding there was such a significant discrepancy between HEDR

16 model prediction for 1946 and the historical measurements of

17 1946. There is no indication that Goble discussed "gamma

18 spectrometry" in his reports or made any assertion the "wet

19 chemistry" method was in fact unreliable. Indeed, if "gamma

20 spectrometry" is far superior to "wet chemistry," what does that

21 say about the even more primitive and less reliable Geiger-

22 Mueller pellet analysis? And if gamma spectrometry is the far

23 superior method, that increases the importance of the vegetation

24 data (historical measurements) taken after introduction of the

25 gamma spectrometry method. In other words, this later data might

26 offer a more accurate comparison of HEDR model predictions and

27 historical measurements. As will be discussed <u>infra</u>, Goble did

28 **ORDER RE SUMMARY JUDGMENT-**   **558**

1   not consider the vegetation data available after 1946 and after

2   the introduction of either the "wet chemistry" or the "gamma

3   spectrometry" method.   (Goble Dep. at pp. 104 and 160).

4   Plaintiffs tender several other arguments pertaining to the

5   purported unreliability of the "wet chemistry" method. They

6   assert the discrepancy between model predictions and historical

7   measurements in the Green Run data is really not a factor of 1.3

8   (as even Goble stated), but is a factor of 3.25. They cite an

9   April 1997 letter from Bruce Napier, the Chief HEDR scientist,

10 who acknowledged an incorrect conversion factor was used with

11 regard to the Green Run data and that "[a]pplication of the

12 correct conversion factor would tend to increase the measured

13 concentrations by a factor of about 2.5."[451]

14   There is still a significant gap between 3.25 for December

15 1949 and the 11.7 discrepancy Goble found for the winter months

16 of 1946 (before application of his wet/dry ratio).[452] Also, any

17 biases in the "wet chemistry" method do not necessarily eliminate

18 the biases existing in the Geiger-Muller pellet analysis which

19 was used to collect the 1946 vegetation data.

20   Plaintiffs point out there were problems with the Green Run

21 environmental monitoring effort and that during the Green Run,

22 the laboratory used for counting environmental samples became

23 contaminated, resulting in spurious measurements. The HEDR

24 Validation Report specifically considered the laboratory

25 _____

    [451]   1.3 x 2.5= 3.25

26

    [452]   Defendants challenge the validity of this wet/dry weight

27 ratio. It is discussed _infra_.

28

ORDER RE SUMMARY JUDGMENT-   559

1 contamination in its determination of how well HEDR model

2 predictions compared with Green Run historical measurements. It

3 still concluded "the model estimates were very close to actual

4 occurrence for this single release event." (HEDR Validation

5 Report at pp. 5.12 and 5.13).

6   Plaintiffs merely identify problems with environmental

7 monitoring associated with collection of the Green Run data and

8 invite speculation as to the extent to which this may have

9 impacted the quality of the Green Run data. In his reports,

10 Goble does not identify problems with laboratory contamination

11 and environmental monitoring during the Green Run as a reason for

12 not considering the Green Run data.

13   Plaintiffs contend defendants misrepresent Goble's

14 statements about the reliability of the "wet chemistry" method.

15 They point out that in his deposition, Goble prefaced his remarks

16 about the advantages of the "wet chemistry" method by stating he

17 was "assuming" the method was working properly. (Goble Dep. at

18 p. 108). In his reports, Goble did not discuss the difference

19 between the various techniques used to measure I-131. In neither

20 his reports or at his deposition did he assert there is any basis

21 for considering the "wet chemistry" technique to have been

22 unreliable as used by Hanford employees, or less reliable than

23 the Geiger-Mueller pellet analysis. In other words, he never

24 said the Green Run data should be ignored because the "wet

25 chemistry" technique was not working the way it was supposed to.

26   Instead, Goble focused on iodine retention time and weather

27 (fog) as a basis for differentiating the Green Run data from the

28 **ORDER RE SUMMARY JUDGMENT-**   **560**

1 Calendar Year 1946 data. Furthermore, if the court has not

2 already said it enough times, any problems with the "wet

3 chemistry" technique are not necessarily going to eliminate

4 deficiencies of the Geiger-Mueller pellet analysis, deficiencies

5 which Goble did not consider in arriving at conclusions about the

6 extent of the discrepancy between HEDR model predictions for

7 Calendar Year 1946 and the vegetation measurements for 1946.

8   In sum, plaintiffs' discussion about the purported

9 unreliability of the "wet chemistry" method is a diversion from

10 the real issue: as a matter of sound scientific methodology,

11 should Goble have considered and quantified the uncertainty

12 involved in the Geiger-Mueller pellet analysis, and should he

13 have attempted to validate his dose estimation model which is

14 based solely on the 1946 vegetation data, with subsequent data

15 produced after the introduction of more reliable measurement

16 techniques, including "wet chemistry" and "gamma spectrometry?"

17 The court believes the answer is "yes he should have."

18

19   **(3) Wet/Dry Conversion Ratio**

20   Goble's vegetation multiplication or correction factor is

21 based in part on an assertion that the sagebrush measured in 1946

22 was wet, while HEDR made its predictions based on dry sagebrush,

23 thus accounting for an even larger difference between HEDR model

24 predictions and the actual measurements of iodine. Goble uses a

25 wet/dry ratio of 2.25 ("the needed adjustment is the ratio of wet

26 weight to dry weight of sagebrush"). The 2.25 ratio is the

27 result of work done by plaintiffs' expert, Dr. Thomas Cochran.

28 **ORDER RE SUMMARY JUDGMENT-   561**

1   Cochran, "Calibration Of The Hanford Environmental Dose

2   Reconstruction Using Vegetation Data," (March 28, 1996), at pp.

3   3-5.[453]

4   Defendants contend the 2.25 wet/dry ratio is erroneous

5   because it was derived based on an assumption that the sagebrush

6   samples taken in 1946 included both "bud sagebrush" (artemisia

7   spinescens) and "big sagebrush" (artemisia tridentata). "Bud

8   sagebrush" has not been identified as one of the species of

9   vascular plants present at Hanford. M.R. Sackschewsky et al.,

10   "Vascular Plants of the Hanford Site," (1992).[454]

11   Plaintiffs acknowledge this is the case. They admit "bud

12   sagebrush" should be removed from the ratio. According to

13   plaintiffs, without "bud sagebrush," the wet/dry ratio is reduced

14   to 1.77. Plaintiffs say Goble included "bud sagebrush" in his

15   calculations because HEDR's Parameters Report[455] listed a

16   wet/dry ratio for "bud sagebrush, browse," in addition to "big

17   sagebrush, browse,""rabbitbrush, browse," and "small rabbitbrush,

18   fresh browse."[456] Thus, although Goble admitted at his

19   deposition that he had not reviewed any documentation to

20   determine the type of vegetation present around Hanford in 1946

21   ────────────────

        [453]   Hereinafter, "Cochran 1996 Report."

22   [454]   Defendants' Ex. 109.

23
        [455]   Snyder, et al, "Parameters Used in the Environmental
24   Pathways and Radiological Dose Modules (DESCARTES, CIDER, and CRD
     Codes) of the Hanford Environmental Dose Reconstruction
25   Integrated Codes (HEDRIC)," (1994). (Defendants' Ex. 97).

26   [456] Plaintiffs' expert, Dr. Thomas Cochran, averaged the
     wet/dry ratios of these four species to come up with the 2.25
27   figure used by Goble. (Cochran 1996 Report at pp. 3-5).
28
     ORDER RE SUMMARY JUDGMENT-   562

1 (Goble Dep. at p. 146), plaintiffs say this does not reflect

2 poorly on Goble's or Cochran's methodology because they were

3 entitled to rely on the listing of "bud sagebrush" in the HEDR

4 Parameters Report.

5   Plaintiffs say another basis for excusing the error is that

6 Cochran and Goble came up with a wet/dry ratio to correct an

7 "admitted" oversight by HEDR in its Validation Report. According

8 to plaintiffs, the problem is that in the validation exercises,

9 HEDR failed to "convert back from dry weight [model] predictions

10 to wet weight [model] predictions to allow for an 'apples to

11 apples' comparison with the wet weight historical data."

12   Defendants do not seem to contest there is a need for a

13 wet/dry ratio. However, they contend that in addition to Goble's

14 figure (2.25) being erroneous, it is also speculative because it

15 fails to consider how much the sample would have dried over the

16 course of one day after it had been picked from the rooted plant.

17   Defendants note that Cochran concluded the time period

18 between collection and counting of vegetation samples was unknown

19 and he "assumed the time period [was] uniformly one day."

20 (Cochran 1996 Report at p. 3). Cochran did this as part of his

21 effort to "correct" the vegetation measurements to account for

22 the decay of I-131 between the time the vegetation was collected

23 and the time the sample was counted. Cochran observed that by

24 assuming a uniform day between collection and counting, this

25 **increased** the measured values by 9%. Id. Defendants argue that

26 whereas plaintiffs are willing to use the one day assumption as a

27 basis for increasing Goble's calibration factors, they are not

28 **ORDER RE SUMMARY JUDGMENT-   563**

1 willing to accept that assumption for the purpose of calculating

2 the wet/dry ratio.   At his deposition, Goble admitted he did not

3 know how much a sagebrush sample would dry over the course of a

4 day.   (Goble Dep. at p. 140).

5   Plaintiffs contend defendants have offered no evidence as to

6 how much a sample would actually dry over the course of a day.

7 The plaintiffs are correct in this regard.   Furthermore,

8 plaintiffs cite evidence that no special efforts were taken to

9 sample dry vegetation and that desert vegetation has a number of

10 mechanisms by which it minimizes water loss.   (Plaintiffs'

11 Response Br. at p. 64, n. 80).   For example, plaintiffs cite Mart

12 1993 wherein it is stated that "vegetation was not artificially

13 dried," but "was counted in its natural condition upon arrival

14 from the field . . . ."   (Mart 1993 at p. 2.2).

15   Plaintiffs' counsel assert there is an additional error in

16 HEDR.   The HEDR Parameters Report lists a value for "big

17 sagebrush, browse," the source of which is M.E. Ensminger, et al,

18 Feeds & Nutrition, 2d Ed. (1990).[457]   The listing in Ensminger

19 1990 is for "Sagebrush, Big, Browse, Stem Cured, Fresh."

20 Plaintiffs assert that because "stem cured" sagebrush is drier

21 (has lower water content) than other varieties of big sagebrush

22 such as "browse" and "fresh," this has the effect of increasing

23 the wet/dry ratio and making Cochran's ratio of 2.25 reasonable,

24 even without including "bud sagebrush" in the calculations.

25   This, however, is not an argument tendered by either Cochran

26

27   [457] Defendants' Ex. 27.

28 **ORDER RE SUMMARY JUDGMENT-   564**

1   or Goble.   Furthermore, it seems almost an afterthought in light

2   of plaintiffs' concession that "bud sagebrush" should not have

3   been included in the ratio.   In other words, it is a belated

4   attempt by counsel to rehabilitate the 2.25 wet/dry ratio.

5   In their reply brief, the defendants contend another factor

6   diminishing the wet/dry ratio is the fact the issue is not the

7   relationship between wet sagebrush and **dry sagebrush**, but between

8   wet sagebrush and **dry pasture grass**.   Converting from I-131

9   concentrations in wet sagebrush to I-131 concentrations in dry

10 pasture grass is necessary for figuring out thyroid dose from

11 milk from a cow which ate pasture grass.

12   In his 1996 report, Cochran offered a couple approaches for

13 making this conversion.   (Cochran 1996 at pp. 14-15).   Based on

14 equations used by HEDR, Cochran came up with monthly ratios of

15 the concentration of I-131 in dry pasture grass to that in dry

16 sagebrush.   Those ratios range from a high of 0.96 in January to

17 a low of 0.69 in June and July.   This means that for January, the

18 concentration of I-131 in dry pasture grass was 0.96 of the

19 concentration in dry sagebrush and for June and July, 0.69 of the

20 concentration in dry sagebrush.   One of Cochran's approaches is

21 to start with his 2.25 wet/dry ratio for comparing the

22 concentration of I-131 in wet sagebrush to that in dry sagebrush,

23 and then multiply that by the monthly ratios of the concentration

24 of I-131 in dry pasture grass versus that in dry sagebrush.   For

25 January, 2.25 is multiplied by 0.96 to produce a figure of 2.15.

26 For June and July, 2.25 is multiplied by 0.69 to produce a figure

27 of 1.56.   Including the sagebrush to grass ratio has the effect

28 **ORDER RE SUMMARY JUDGMENT-   565**

1   of **reducing** the I-131 dose from milk from a cow which ate pasture

2   grass.[458]   According to defendants, Goble did not include the

3   sagebrush to pasture grass factor in his calibrations, thereby

4   increasing his dose estimates by 22%.[459]

5   The court notes this is not an argument which defendants

6   specifically tendered in their opening brief. On the other hand,

7   while plaintiffs seek to file a surreply, it is not clear that

8   this is one of the arguments to which they seek to respond. As

9   far as the court can discern, the plaintiffs' response brief

10 contains no discussion about Goble reducing doses due to the

11 sagebrush to pasture factor. At his deposition, Goble testified

12 he would use HEDR's analysis of the sagebrush-to-grass adjustment

13 (i.e. the monthly ratios calculated by Cochran using HEDR's

14 equations). Goble's testimony indicates he had not included that

15 adjustment in his "calibration" of the HEDR model:

16   The HEDR model predicts different concentrations
            for pasture grass than it does for sagebrush, and
17   I **would** use their analysis of the differences between
            pasture grass and sage brush to make that transition.
18
(Goble Dep. at p. 120)(Emphasis added). Defendants say this is
19
    yet another example of Goble being selective about his choice of
20
    data in an effort to increase dose estimates.
21
        Finally, defendants also point to deposition testimony from
22
Cochran acknowledging "considerable uncertainty" in the wet/dry
23

24   [458] Obviously, the figures are even lower if one starts out
with a wet/dry ratio of 1.77 for comparing the concentration of
25 I-131 in wet sagebrush to that in dry sagebrush.

26   [459] The average monthly ratio of the concentration of I-131
in dry pasture grass to that in dry sagebrush is 0.82. 2.25 x
27 0.82 equals 1.845. 2.25/1.845= 1.22 or 22%.

28
    ORDER RE SUMMARY JUDGMENT-   566

ratio and that it would be "useful" to "see if we can get a more

accurate estimator for the wet to dry ratios, both the sagebrush

and pasture grass and sagebrush to pasture grass and so forth."

(Cochran Dep. at p. 292).   Cochran expressed a similar sentiment

in his 1996 report, stating "[a]dditional research and analysis

could shed further light upon what constitutes the best estimate

of this ratio."   (Cochran 1996 Report at p. 23).   Asked what type

of additional research should be done, Cochran responded:

> Well, you are asking me for the protocol of
> that type of research, and I would not- the
> thing I would not do is take an instant
> opinion of somebody like myself, who is unfamiliar
> with the biological parameters that would be
> involved and the various other- and also, I
> would want to get other experts in designing
> protocol for such an experiment, who are
> familiar with other aspects that would enter
> into that type of research effort.

(Cochran Dep. at p. 296).

In a footnote in their response brief, plaintiffs say that

although Cochran indicated there was "considerable uncertainty"

in the wet/dry ratio, he also noted that it was "actually small

when compared to the uncertainty in the dose estimates using the

HEDR model."   Plaintiffs, however, do not cite to anything from

Cochran's reports or depositions where such a statement can be

found.

The wet/dry ratio is a troublesome issue.   Defendants appear

to concede some sort of ratio is necessary, although they have

not proposed a particular figure.   It appears there is a

legitimate dispute about how dry the samples would have been at

the time of counting.   As such, the court concludes there is a

ORDER RE SUMMARY JUDGMENT-   567

1 genuine issue of material fact about the proper wet/dry ratio.

2   The question remains, however, whether the ratio proposed by

3 Goble and Cochran is methodologically sound. Plaintiffs readily

4 acknowledge "bud sagebrush" should not have been included in the

5 ratio calculation. However, because "bud sagebrush" is listed in

6 HEDR's Parameters Report, the court is hesitant to say that

7 Cochran's inclusion of it in his ratio calculation makes that

8 calculation per se methodologically unreliable. Cochran's

9 statements about the uncertainty of his ratio are refreshingly

10 candid. Those statements cannot be construed as a concession

11 that his ratio is unscientifically reliable. In sum, the court

12 cannot say the modified 1.77 ratio, excluding bud sagebrush, is

13 scientifically unreliable and therefore, inadmissible.[460]

14   On the other hand, the wet/dry ratio is but one piece of

15 **Goble's** dose estimation analysis. Goble's apparent failure to

16 consider and include in his calibration analysis a sagebrush-to-

17 pasture adjustment exhibits a "selectivity of data" which is

18 inconsistent with the scientific data. Combining this with his

19 failure to adequately and specifically consider the uncertainty

20 of the Calendar Year 1946 vegetation data set due to limited

21 measurement techniques available at the time, raises serious

22 questions about the soundness of his methodology.

23 //

24 //

25

    [460] The court need not consider plaintiffs' argument that
26 2.25 is probably a reasonable figure taking into account big
sagebrush in a "stem cured" state. This argument has not been
27 advanced by any expert.

28

ORDER RE SUMMARY JUDGMENT-   568

(4) Milk Concentration Data/September 1963 PUREX Release

Another of the data sets upon which HEDR relied in an effort

to validate model predictions is that pertaining to the

accidental release of approximately 72 curies of I-131 from the

PUREX Plant in September 1963. The available data includes: 1)

air measurements taken at daily intervals at Benton City,

Richland, and Kennewick, as well as 18 onsite locations; 2)

measurements of pasture grass taken daily at two farms in cell

468[461] (Farm A and Farm B) and sporadically at numerous other

locations; 3) milk measured at two farms in the Benton City area

(Farm A and Farm B) and at the local creameries; and 4) thyroid

counts taken on two children who consumed milk from a cow in the

backyard of one of the farms (Farm B). (HEDR Validation Report

at pp. 6.1 and 6.3).

At both Farm A and Farm B, individual family cows were on

pasture and providing milk to residents of the farms. The milk

was for personal consumption. HEDR model predictions of the I-

131 concentration in the milk at Farm A and the historical

measurements thereof are "essentially equal." HEDR model

predictions of I-131 concentration in the milk at Farm B

underestimates by "about a factor of 3" the historical

measurements of said concentration "when extrapolated over the

entire period." (HEDR Validation Report at p. 6.4). In other

words, milk concentration (historical data) exceeds the estimate

(HEDR model prediction) by "about a factor of 3." HEDR

---

[461] Each HEDR study area is comprised of individual
geographic cells.

ORDER RE SUMMARY JUDGMENT—   569

1 considered these to be favorable comparisons between model

2 predictions and historical measurements.

3   Defendants say Goble ignored this milk concentration data

4 even though he acknowledged that for I-131 dose reconstruction

5 purposes, it was more important for the HEDR model to accurately

6 predict milk concentrations than pasture grass concentrations.

7 (Goble Dep. at p. 160).[462] According to defendants, the dose

8 estimates generated by Goble's method (Goble's predictions) would

9 "fail miserably" if compared to this milk concentration data.

10   For Farm A, HEDR model predictions and the historical

11 measurements of I-131 concentration in the milk are "essentially

12 equal." Under Goble's method, an 8.7 vegetation multiplication

13 or correction factor would be applied to HEDR's dose estimate

14 (model prediction).[463] Then, say defendants, Goble's method

15 would require the HEDR dose estimate (model prediction) to be

16 increased by a factor of 20 to account for the source term

17 multiplication factor. The source term factor comes from

18 plaintiffs' source term experts, the Herrmanns.[464] Defendants

19 acknowledge the Herrmanns only provided release estimates through

20 1960, but contend Goble testified his method could be extended

21 beyond 1960. However, defendants do not say where this testimony

22 _____

[462] The milk pathway is the most significant pathway for I-
23 131 exposure to the iodine-sensitive thyroid gland.

24   [463] 8.7 factor applicable for the months of March through
November. The PUREX release occurred in September 1963.

25
[464] According to defendants, the Herrmanns estimate 47,416
26 curies of I-131 were released between 1955-1960, while HEDR's
estimate is 2,345 curies. 47,416 divided by 2,345 equals 20.2.
27 Plaintiffs do not contest the accuracy of these figures.

28
ORDER RE SUMMARY JUDGMENT-   570

1   can be found.

2   Defendants assert Goble's method would overpredict the milk

3   concentration at Farm A by a factor of 174 (8.7 vegetation factor

4   x 20 source term factor). For Farm B, defendants indicate

5   Goble's dose estimation method would overpredict milk

6   concentration by a factor 58. The factor of 58 comes from

7   dividing 174 by 3, the amount by which HEDR says its model

8   predictions underestimate historical measurements. In other

9   words, while HEDR underpredicts by a factor of 3, Goble would

10   overpredict by a factor of 58.

11   Plaintiffs acknowledge Goble did not use the milk

12   concentration data from the 1963 PUREX release, but they claim he

13   had valid reasons for not doing so. Plaintiffs assert that like

14   the Green Run data set, the 1963 PUREX release data set is not a

15   "time-sequence set" and therefore, not helpful for determining

16   average concentrations of I-131 over longer periods of time. Put

17   another way, the PUREX release is more on the order of a single

18   release akin to the Green Run.

19   Goble's 1995 report states the following about the PUREX

20   data:

21   Figure 3.3a and 3.3b appear to show that the
             HEDR model predicted early concentrations in
22   pasture grass and milk reasonably well for
             Farm A and they under predict the early
23   concentrations in Farm B by a factor of 3 to 4
             for the early period after the release. **As in**
24   **the Green Run, the tendency is for the**
             predictions to fall off too rapidly for later
25   **periods.**

26   (Goble 1995 Report at p. 61). (Emphasis added).

27   Rather than focus on the milk concentration data, Goble's
28
     ORDER RE SUMMARY JUDGMENT-   571

1 reports (and plaintiffs' counsel) focus: 1) on the comparison of

2 HEDR model predictions with pasture grass contamination data; and

3 2) on the comparison of HEDR model predictions with the thyroid

4 dose burdens on the two children consuming milk at Farm B.

5   Footnote 39 at p. 61 of Goble's 1995 report states the

6 following about the pasture grass and milk concentration

7 predictions:

> There is . . . an internal inconsistency in
> these predictions. Either the predicted
> concentrations of I-131 in pasture grass are
> expressed in terms of dry weight of the grass-
> in which case they significantly underestimate
> the measurements even at Farm A, since the Soldat
> data is for wet weight (estimated to be 80%
> of the weight)(Soldat 1965) Table 2, p. 1012[465];
> or they have been correctly adjusted for moisture,
> but then the prediction of I-131 in the milk is
> inconsistent with the assumptions in (Snyder 1992)
> and (Ikenberry 1992).

As noted, Goble eventually confirmed HEDR's predictions of

I-131 were based on the dry weight of the vegetation, whereas

measurements were based on wet weight. Goble concluded "this

error means that the predictions are an even smaller fraction of

the measurements, probably by more than a factor of two." (Goble

1996 Report at p. 15). The issue of wet/dry ratio in terms of

model predictions of I-131 concentration based on dry sagebrush

versus historical measurements based on wet sagebrush has already

been discussed. Goble used a wet/dry ratio of 2.25 for that

purpose. As between **wet pasture grass and dry pasture grass**,

plaintiffs assert the wet/dry ratio is even higher.

---

[465] Soldat, "Environmental Evaluation of an Acute Release of I-131 to the Atmosphere," Vol. 11 **Health Physics** (1965). Hereinafter referred to as "Soldat 1965."

ORDER RE SUMMARY JUDGMENT-   572

1   Plaintiffs note that HEDR's Parameters Report provides a

2 dry-to-wet weight ratio of 0.35 for "Pasture." (HEDR Parameters

3 Report at p. 6.65). That translates to a wet-to-dry weight ratio

4 of 2.85 (1 divided by 0.35). Plaintiffs assert that "if this

5 ratio reasonably represented the wet-to-dry ratio of the

6 vegetation sampled in and around the September 1963 PUREX

7 incident, then HEDR predictions should be reduced by a factor of

8 2.85 to obtain a wet-weight HEDR prediction for accurate

9 comparison to wet-weight vegetation results." However, because

10 Soldat 1965 estimated 80% of the vegetation weight was water,

11 plaintiffs say a "wet-to-dry ratio of 5 may be more reasonable

12 for the vegetation sampled during this period" (1 divided by

13 0.20 equals 5). According to plaintiffs, Dr. Goble "comes to the

14 obvious conclusion [that] HEDR predictions for pasture grass for

15 the 1963 PUREX incident should be reduced by a factor of 5 to

16 allow for an accurate comparison."

17   Interestingly, plaintiffs do not cite where in any of his

18 reports or in his deposition Goble actually ran through the

19 numbers as such and arrived at this specific conclusion. It

20 appears plaintiffs' counsel simply seized upon footnote 39 in

21 Goble's 1995 report and extended Goble's analysis on his behalf.

22   Defendants do not attempt to rebut this conclusion or the

23 assumptions on which it is based (80% of the vegetation weight

24 was water). Instead, they argue the focus should remain on the

25 milk concentration data, rather than the pasture grass

26 concentration data. Goble acknowledged it was more important for

27 the HEDR model to accurately predict milk concentrations than

28 **ORDER RE SUMMARY JUDGMENT-**   **573**

1   pasture grass concentrations. Goble also stated **if the predicted**

2   **concentrations of I-131 in pasture grass had been correctly**

3   **adjusted for moisture,** only then would the prediction of I-131 in

4   the milk be inconsistent with the assumptions in Snyder 1992

5   (HEDR Parameters Report) and Ikenberry 1992. Goble concluded the

6   concentrations in I-131 had **not been correctly adjusted for**

7   **moisture.** Logically then, Goble can no longer assert HEDR's

8   prediction of I-131 in milk is inconsistent with any assumptions

9   in Snyder 1992 or Ikenberry 1992.

10   The plaintiffs try to show Goble's estimates are "validated"

11   by the 1963 milk concentration data. Plaintiffs contend

12   defendants errantly assume Goble would include a source term

13   correction factor of 20 for the September 1963 PUREX release.

14   Because the Herrmanns did not provide any release estimates for

15   after 1960, plaintiffs say the defendants are engaging in

16   "unscientific speculation" that Goble would include a source term

17   correction factor. Plaintiffs say that "[s]caling to a single

18   release event is obviously inappropriate, and constitutes

19   'selective use' of data by the defendants' lawyers."

20   According to plaintiffs, the "appropriate Goble correction

21   factor for milk concentration for [the PUREX] event" is only the

22   8.7 vegetation correction factor for the summer months (March

23   through November). Based on this, plaintiffs admit Goble's

24   estimates would overpredict the milk concentration at Farm A by a

25   factor of 8.7 (as opposed to 174), and at Farm B by a factor of

26   2.9 (8.7 divided by 3). The average overprediction is 5.8 for

27   both farms (11.6 (8.7 + 2.9)/2 = 5.8).

28   **ORDER RE SUMMARY JUDGMENT-   574**

1   However, plaintiffs contend the "feed[466] to milk transfer

2 ratio for the cows on these farms is significantly lower than the

3 average through the early 1960s." Plaintiffs cite Cochran's 1996

4 report wherein he found that the average ratio of the

5 concentration of I-131 in milk to that in grass was actually

6 0.183 based on sampling of five dairy farms during ten separate

7 months in 1961-62. On the other hand, the data from the two

8 farms (Farm A and B) studied following the PUREX release of

9 September 1963 revealed an average ratio of 0.07. Cochran noted

10 these two results (0.183/0.07) differed by a factor of 2.6.

11 Plaintiffs assert that if the cows at Farm A and B following the

12 1963 PUREX release exhibited an "average feed to milk transfer

13 ratio, Dr. Goble's prediction would be high by only a factor of

14 2.2 and HEDR would be low by a factor of 5.2."

15   Plaintiffs' argument appears to be that milk concentrations

16 in 1963 would have actually been lower and therefore, there is

17 not as great a gap between Goble's predictions and the milk

18 concentration data for 1963 (5.8/2.6 = 2.2). On the other hand,

19 because milk concentrations are lower for 1963, plaintiffs say

20 that widens the discrepancy between HEDR's predictions and the

21 milk concentration data for 1963.[467]

22

23   [466] Pasture grass.

24   [467] The 5.2 estimated underprediction for HEDR dose
estimates is derived by averaging the underprediction for Farm A

25 and Farm B. For Farm A, there is no underprediction. For Farm
B, plaintiffs say there is a 3 to 4 underprediction. Plaintiffs

26 take the higher figure of 4 and divide it by 2 to get an average
underprediction of 2 for both Farm and Farm B. This average of 2

27 is then multiplied by 2.6 to arrive at 5.2.

28
ORDER RE SUMMARY JUDGMENT-   575

1   Defendants contend that if the plaintiffs are not using a

2 source term multiplier based on the Herrmanns' release estimates,

3 it means plaintiffs are accepting the historical measurement of

4 **releases** from the September 1963 PUREX incident. The historical

5 measurement is the amount measured by the Hanford stack sampling

6 system: 72 curies of I-131. (HEDR Validation Report at p. 6.3).

7   Defendants assert plaintiffs' "uncritical reliance" on the

8 stack sampling data is contrary to the Herrmanns' source term

9 analysis for 1948 to 1960 which concluded stack sampling records

10 were not reliable. According to the Herrmanns:

11   The ideal method for determining the iodine-131
         release fraction would take the ratio of the
12   measured iodine-131 released through the stack,
         and the iodine-131 processed. **This method is not**
13   **applicable because reliable values of iodine**
         released to the stacks for Hanford are not available.
14
(Herrmann Report at Section 2.2, p. 11)(Emphasis added).
15
      Defendants argue that if the stack data is unreliable, Goble
16
cannot use it to validate his methodology. If the data is
17
reliable, then the defendants contend the Herrmanns' source term
18
analysis is of no value. Defendants say it is scientifically
19
inappropriate for Goble to rely on the Herrmanns' source term
20
analysis to determine dose estimates for the period of 1948-1960,
21
but for the purpose of "validating" dose estimates, ignore the
22
Herrmann's analysis and rely on stack sampling data which the
23
Herrmanns stated was not reliable.[468]
24
      Apparently because I-131 releases tailed off so
25
_____

26   [468] There is a question whether Goble himself actually
undertook any type of validation process, or whether plaintiffs'
27 counsel tried to do it for him. This is discussed _infra_.

28
ORDER RE SUMMARY JUDGMENT-   576

1 significantly after 1960, plaintiffs determined it was not worth

2 the Herrmanns' time to analyze such releases. Therefore,

3 plaintiffs have opted not to provide I-131 release estimates

4 beyond 1960. At oral argument, plaintiffs' counsel conceded they

5 could not argue "on the current state of the record, that there

6 were sufficient iodine releases solely after 1960 for a person

7 that wasn't there before 1960 or wasn't born before 1960 for that

8 person to be affected." (Tr. of Oral Argument at pp. 65-66).

9   It is not clear what the Herrmanns would say about the

10 quality of stack sampling in 1963. It is also true that the

11 PUREX release was confined to a single month. Therefore, there

12 appears to be speculation as to what, if any, source term

13 multiplication factor plaintiffs would propose for any dose

14 estimates Goble might produce for September 1963. As noted

15 above, defendants do not provide any citation where Goble stated

16 his method could be extended beyond 1960.

17   At the same time, if the plaintiffs are not going to bother

18 with dose estimates beyond 1960, they should not be able to use

19 1963 historical data in an attempt to validate Goble's iodine

20 dose estimates which apparently go no further than 1960.

21 Validation of Goble's dose estimates can only be based on

22 historical data from 1960 and earlier.[469] As such, plaintiffs'

23 discussion about the difference in feed to milk transfer ratio

24 between 1961-1962 and 1963 is of no consequence insofar as

25 _____

26 [469] HEDR provides dose estimates beyond 1960. Therefore,
    the PUREX release can be used in an effort to validate its

27 estimates.

28

ORDER RE SUMMARY JUDGMENT-   577

1   validation.

2   In any event, Goble never said anything about how the feed

3   to milk transfer ratio would validate his dose estimates. That

4   discussion and analysis is entirely the doing of plaintiffs'

5   counsel. Furthermore, the court points out that Cochran, who

6   discussed the discrepancy between the feed to milk transfer ratio

7   as between 1961-62 (O.183) and 1963 (0.07), stated that

8   "[a]rguably, the lower figure [0.07] could be discarded."

9   Indeed, Cochran settled on a combined ratio of 0.15 for 1961-62

10   and 1963, giving the ratio of 0.183 three times the weight of the

11   0.07 ratio. (Cochran 1996 Report at pp. 15-16).[470]

12   In sum, the court cannot find it was scientifically improper

13   for Goble to ignore the 1963 milk concentration data. This is

14   because such data cannot be of any assistance in validating his

15   dose estimates which apparently do not go beyond 1960. At the

16   same time, neither plaintiffs or Goble have identified anything

17   in the milk concentration data showing that it somehow

18   invalidates HEDR's model predictions for September 1963.

19   Consequently, we are still left with nearly an identical match

20   for Farm A and an underprediction of about 3 for Farm B.

21   According to the HEDR Validation Report, "[c]omparisons of

22   estimated versus historical measurements must be made, and the

23   general objective is that the measurements and estimates compare

24   to within a factor of 3." (HEDR Validation Report at p. 1.6).

25   _____

26   [470] It appears the result would be to substantially reduce,
     if not eliminate, the 2.6 feed to milk ratio which plaintiffs
     employ to reduce Goble's overprediction of I-131 concentration in
27   milk.

28

ORDER RE SUMMARY JUDGMENT-   578

1 HEDR considered anything within a factor of "3" to constitute a

2 favorable comparison between model predictions and historical

3 measurements.

4   Apparently realizing he could not take HEDR to task based on

5 the September 1963 milk concentration data, Goble went on to

6 criticize HEDR about its conclusions regarding consistency

7 between its model predictions and the thyroid dose burdens on the

8 two children who drank milk from a cow pastured on Farm B.

9   Thyroid dose burdens were measured in October 1963.

10 According to the HEDR Validation Report, the parents of the

11 children indicated that at the time of the exposure, the 4-year-

12 old boy was consuming about 1 gallon of milk per day from the

13 family cow, while his 8-year-old sister was consuming about 1

14 quart per day. HEDR estimated the dose in 1963 for the boy was

15 about 35 mrad to the thyroid, and for the girl 25% of that amount

16 or 9 mrad. The Validation Report concluded "[t]he measured doses

17 fall well within the ranges estimated by the HEDR models using

18 the 1963 dose conversion factor." (HEDR Validation Report at p.

19 6.7).

20   In his 1995 report, Goble leveled this criticism at HEDR:

21   Both Soldat [1965] and the validation report
            assert they find agreement between the dose
22   they estimate and the results of the single
            measurement on the thyroid made in October,
23   though the assumptions made are different. Soldat
            assumes contamination in milk as measured at
24   Farm B and consumption of milk by the child
            of about 1 liter per day, in contrast to the
25   parents' assertion that the child drank a gallon
            a day. The validation report uses the one gallon
26   figure, but bases its estimates on the predictions
            which were a factor 3 to 4 lower than the measure-
27   ments on Farm B. Thus Soldat and the validation

28 **ORDER RE SUMMARY JUDGMENT-   579**

1   report estimate similar doses, but with canceling
      contradictory assumptions. In neither case

2   does the estimate relate very closely to the single
      measurement in the thyroid, since most of the

3   radioactivity still remaining was almost certainly
      consumed in October- unless the child stopped

4   drinking milk for those weeks.

5 (Goble 1995 Report at p. 61).

6   The issue here is the soundness of Dr. Goble's dose

7 estimation methodology, not the soundness of HEDR's methodology.

8 Goble's criticism is irrelevant, notwithstanding its seeming

9 validity. Plaintiffs attempt to validate Goble's dose estimation

10 methodology based on the thyroid dose burden data and Goble's

11 criticism of what he alleges are "canceling contradictory

12 assumptions" between Soldat and the HEDR Validation Report.

13 However, plaintiffs cannot use the 1963 PUREX data for validation

14 purposes if they are not going to bother with any iodine dose

15 estimates for after 1960.

16

17   **(5) Validation of Goble's Dose Estimation Methodology**

18   Defendants contend Goble's dose estimation methodology is

19 scientifically unreliable because he made no attempt to validate

20 his own dose estimates against historical measurements. They

21 assert that plaintiffs' counsel simply engage in a "hypothetical

22 validation" on Goble's behalf.

23   Goble acknowledged that "comparisons of models with data are

24 standard practice." (Goble Dep. at pp. 74-75). Indeed, Goble's

25 dose estimation method is based on criticism of HEDR's

26 validation, in particular the discrepancy between HEDR model

27 predictions and the Calendar Year 1946 vegetation data.

28 **ORDER RE SUMMARY JUDGMENT-** **580**

1   Plaintiffs acknowledge validation is necessary, but they

2 note that Goble testified "it is good practice to compare dose

3 reconstruction models to **good data.**" (Goble Dep. at p.

4 63)(Emphasis added). This goes back to plaintiffs' arguments

5 that the Green Run data and PUREX Release data are not "good

6 data" for the purpose of determining "long term average

7 measurements during continuous releases."   While this data may

8 validate model predictions for December 1949 and September 1963,

9 plaintiffs suggest it is not suitable for validating model

10 predictions based on "continuous releases" from day to day

11 operations. Of course, plaintiffs say this is why Goble used

12 the Calendar Year 1946 vegetation data set for calibration

13 purposes.

14   At his deposition, Goble was asked whether in his reports he

15 had compared the predictions of **his dose method** to any of the

16 vegetation data or other environmental data summarized by HEDR,

17 other than the Calendar Year 1946 data set.   Goble's response

18 was:

19   In my report, I indicate the **general consistency
           or inconsistency** of my calibration with . . .
20   the various data sets in the [HEDR] validation
           report. So there is a comparison to those
21   various data sets. **But other than that, no.**

22 (Goble Dep. at p. 94)(Emphasis added). Goble stated that because

23 his method was essentially the same as HEDR's method, with the

24 exception of a "scaling factor," the comparison to the validation

25 data sets was "in effect" the same comparison. (Id. at p. 95).

26   Goble acknowledged he did not use any of the environmental

27 data other than the data collected and provided by HEDR. (Id. at

28
     ORDER RE SUMMARY JUDGMENT-   581

1   p. 96). This other data includes vegetation data for 1945 and

2   1947, the 1948-51 vegetation data set as a whole, and vegetation

3   data for after 1951.   (Goble Dep. at pp. 97, 103 and 104).

4   While in his reports Goble may have indicated the "general

5   consistency or inconsistency" of his calibration with the data

6   sets included in the HEDR Validation Report, he certainly did not

7   engage in the specific validation analysis contained in the

8   response brief of plaintiffs' counsel. This is not surprising if

9   it is indeed true that Goble (and not just plaintiffs' counsel)

10   considered HEDR's validation data sets (other than the Calendar

11   Year 1946 set) not "representative of long term average

12   conditions" and therefore, inappropriate for validating Goble's

13   predictions. According to plaintiffs, **"even** if these data sets

14   were assumed to be representative of long term average

15   conditions, the comparisons between Dr. Goble's predictions and

16   the environmental data **would** not indicate any 'flaws' in Dr.

17   Goble's method."

18   Plaintiffs' counsel go through the data sets in the HEDR

19   Validation Report- the April 13, 1946 Footprint Data, the

20   December 1949 Green Run Data, and the September 1963 PUREX Data-

21   in an effort to show Goble's predictions stack up well with that

22   data.[471] The court has already discussed this effort in regard

23   to the milk concentration data pertaining to the September 1963

24   PUREX release. Because the plaintiffs and Goble are not offering

25   _____

26   [471] Plaintiffs acknowledge Goble's predictions should not be
     compared to the Calendar Year 1946 vegetation data set which he
27   used as the basis for his "calibration."

28   **ORDER RE SUMMARY JUDGMENT-   582**

1 dose estimates beyond 1960, they cannot use 1963 data in an

2 attempt to validate those dose estimates.

3

4   **(a)   April 13, 1946 Dispersion/Deposition Footprint**

5   This data was derived from sagebrush samples taken in a one

6 day period from Ellensburg to Ritzville, Sprague to Spokane, and

7 Umatilla to The Dalles, Oregon. HEDR used this data set for

8 validation purposes because the samples were taken during

9 "growing season" conditions and because it is during growing

10 season that contamination of plant products is most important to

11 dose.   (HEDR Validation Report at p. 4.1).

12   Defendants do **not** contend Goble should have considered this

13 data in his calibration analysis. This makes sense since I-131

14 in these samples was measured by the same technique (Geiger-

15 Muller) which defendants allege injects significant uncertainty

16 into the accuracy of the I-131 measurements which are part of the

17 Calendar Year 1946 data set.

18   According to plaintiffs, Goble did not use the "footprint"

19 data set because, like the Green Run and the PUREX release, it

20 "does not constitute a long-term average indication of iodine

21 contamination on vegetation." Nevertheless, assuming it would be

22 representative of long-term average conditions, plaintiffs'

23 counsel contend the "footprint" data "validates" Goble's dose

24 estimation, while revealing that HEDR significantly underpredicts

25 the I-131 measurements for April 13, 1946.

26   In his 1995 report, Goble commented that a comparison of the

27 total predicted by HEDR against the measured average

28 **ORDER RE SUMMARY JUDGMENT-   583**

1 concentration of iodine on the vegetation from the footprint

2 data, shows the model underpredicting by a factor of three.

3 (Goble 1995 Report at p. 57). Cochran came to the same

4 conclusion: "The sum of the measured median (wet weight) values

5 is three times greater than the sum of the HEDR estimated (dry

6 weight) values . . . ." (Cochran 1996 Report at p. 6). However,

7 Goble concluded the factor was actually closer to four because it

8 was appropriate to employ a "symmetric comparison." Goble

9 described this as "an unbiased adjustment for the effect of the

10 detection limits" involving comparison of "predictions and

11 measurements whenever either the prediction or the measurement

12 exceeds the measurement limit." According to Goble, HEDR had

13 employed an "asymmetric comparison" comparing measurements

14 against predictions when the predictions are large and

15 discovering the average comparison is closer. (Goble 1995 Report

16 at p. 57).

17   Plaintiffs assert that with regard to the "footprint" data,

18 HEDR underpredicts on average by a factor of nine (9). This is

19 derived by multiplying the factor of four by a wet dry ratio of

20 2.25 (4 x 2.25 = 9). Plaintiffs note that Goble's vegetable

21 correction factor "for the Richland area for the month of April

22 is 8.7."[472] They assert Goble's model predictions are 97%

23 consistent with the footprint data for the Richland area (8.7/9 =

24 0.97).

25   Defendants contend plaintiffs' calculations are in error.

26 _____

27 [472] 8.7 is Goble's vegetation correction factor for the
months of March through November.

28 **ORDER RE SUMMARY JUDGMENT-**   **584**

1 Defendants start out with a HEDR underprediction of three (3),

2 based on Cochran's report. Defendants do not, however,

3 specifically quibble with Goble's assertion that the proper

4 factor to start with is four (4). Defendants say the wet/dry

5 conversion factor should only be 1.77 instead of 2.25 due to the

6 omission of bud sagebrush from the calculation. Defendants say a

7 sagebrush-to-pasture adjustment of 0.76 is also necessary.

8 (Cochran 1996 Report at p. 14). The result, according to

9 defendants, is that HEDR's underprediction is approximately a

10 factor of 4. (3 x 1.77 x 0.76 = 4.04). Starting out with Goble's

11 factor of 4 (derived by a symmetric comparison as opposed to an

12 asymmetric comparison), the final result would be an

13 underprediction by a factor of approximately 5.4 (4 x 1.77 x

14 0.76). However, it is important to keep in mind that HEDR's

15 underprediction may not be as significant considering the

16 uncertainty of the measurement technique used in 1946 (i.e.

17 Geiger-Mueller measured all beta-emitting radionuclides and

18 hence, there is a possibility that there was not as much I-131 as

19 reported).

20   Defendants contend Goble's method would overpredict by a

21 factor of 2.9, however, the court's math says the overprediction

22 would be 2.15 (8.7/4.04). With Goble's modification (i.e. using

23 the factor of four), it would be even less: 8.7/5.4 = 1.6.

24 Plaintiffs note this is just for the Richland area and that

25 application of Goble's distance correction factor for areas

26 further away from Hanford would reduce the vegetation correction

27 factor and in turn, further narrow any discrepancy between

28

ORDER RE SUMMARY JUDGMENT-   585

1   Goble's predictions and historical measurements.

2   While the discrepancy between Goble's predictions and the

3   1946 "footprint" measurements may not appear significant, it must

4   be pointed out again that limitations in the measurement

5   technique used at the time raises the legitimate and distinct

6   possibility there was less iodine on the sagebrush than reported.

7   Hence, Goble's overprediction could well be greater than these

8   figures portray. It all comes back to the issue of whether Goble

9   should have quantified the uncertainty pertaining to the 1946

10   measurement technique[473] and incorporated it as part of his

11   calibration analysis.

12

13   **(b)   Green Run- December 1949**

14   Goble reported "[t]he comparison of average [iodine]

15   concentrations shows that the model only underpredicts in this

16   case by a factor of 1.3." (Goble 1995 Report at p. 57).

17   Plaintiffs say that this factor should be increased by 2.25 to

18   account for the wet/dry ratio, and then further increased by a

19   factor of 2.5 to account for an additional error in the Green Run

20   validation exercise. Bruce Napier, Chief HEDR Scientist and lead

21   author of the HEDR Validation Report, informed plaintiffs'

22   counsel that measured concentrations of iodine should be

23   increased by a factor of 2.5 with regard to the Green Run.[474]

24   _____

25   [473] The uncertainty of Geiger-Mueller technique versus the
"wet chemistry" technique subsequently developed.

26

27   [474] In 1949, the "wet chemistry" technique was being used to
measure iodine on vegetation.

28

ORDER RE SUMMARY JUDGMENT-   586

1   (Napier April 1997 Letter at pp. 2-3). The result, say

2   plaintiffs, is that HEDR underpredicts Green Run iodine

3   measurements on average by a factor of 7.3 (1.3 x 2.25 x 2.5).

4   Plaintiffs acknowledge Goble overpredicts by about a factor

5   of 7 when the Herrmann iodine source term estimate is considered

6   ($1/2.9^{475}$ x 26.3 (Goble winter correction factor) x 0.74 (source

7   term correction factor) = 6.7). However, this is reduced

8   substantially when one takes into account Napier's concession

9   that the measured concentrations of iodine during the Green Run

10 should be increased by a factor of 2.5. The result is an

11 overprediction by a factor of 2.7 (6.7/2.5).

12   Plaintiffs contend that because weather conditions during

13 the Green Run were specifically selected to avoid iodine

14 deposition enhancing conditions such as fog, Goble's "summer

15 correction factor" of 8.7 **"may** be more appropriate for the Green

16 Run comparison than his winter correction factor of 26.3." Goble

17 himself, however, says no such thing and this is utter

18 speculation by plaintiffs' counsel. If the summer correction

19 factor of 8.7 was incorporated, plaintiffs say that Goble's

20 predictions for December 1949 would almost exactly match the

21 historical measurements for that period of time.

22   Defendants contend HEDR, at most, would underpredict by a

23 factor of 5.5, taking into account Napier's concession of a 2.5

24 correction factor. Defendants do not contest application of this

25 2.5 correction factor (1.3 x 1.77 (wet/dry ratio without bud

26 _____

27   [475] 1.3 x 2.25 = 2.9.

28 **ORDER RE SUMMARY JUDGMENT**-   **587**

1   sagebrush) x 0.95 (December sagebrush-to-pasture adjustment[476])

2   x 2.5= 5.46). On the other hand, defendants say Goble would

3   overpredict by a factor of 3.6 using the Herrman source term

4   correction factor (1/5.46 x 26.3 x 0.74), to a factor of 6.3

5   using the Klementiev source term correction (1/5.46 x 26.3 x 1.32

6   source term correction factor).[477]

7   It is important to keep in mind that it is Goble's

8   calibration and not HEDR that is the subject of defendants'

9   motion in limine. Thus, while HEDR may well suffer from some

10   deficiencies and errors, that does not necessarily make Goble's

11   work any more scientifically reliable. Goble did not perform the

12   validation effort described above. Plaintiffs' counsel did that

13   work. Indeed, plaintiffs and Goble would just as well not pay

14   any attention to the Green Run data based on their assertion that

15   it is not representative of "long-term" releases from daily

16   operations. Defendants' calculations are reasonable and accurate

17   and show a significant overprediction on Goble's part regarding

18   the Green Run.

19

20   **(c)   Other Data**

21   Defendants contend Goble's methodology is not scientifically

22   reliable because of his failure to consider available vegetation

23   data from 1945 and 1947, 1948-1951, and beyond 1951. This is

24   _____

25   [476] Cochran 1996 Report at p. 14.

26   [477] Defendants would assert the overprediction is due to
     Goble's use of the Calendar Year 1946 data set when I-131 was
27   measured using the gross beta technique.

28
ORDER RE SUMMARY JUDGMENT-   588

1 especially so, say defendants, because of the improvement in

2 measurement techniques which occurred after 1946.

3   Goble was asked at his deposition why he did not use any

4 data other than the data sets HEDR used for validation purposes.

5 His response was:

6   Well, there are two reasons. Really, three
           reasons. One is that HEDR had done a very
7   thorough review and analysis, and it was
           certainly convenient to be able to work
8   with that.

9   The second reason is that- it was reasons of
           limitations of time and resources; that is
10   a lot of work to not only compile, but analyze
           and assess the quality of the representativeness
11   of and so on, data sets from the start, so that
           would have been a big task.

12
           And the third reason, relating to the second,
13   is that there is a certain danger of selectivity
           when you do this that you pick out one piece of
14   or one small data set, if you are not doing a
           comprehensive search, and this way, I am at
15   least relying on someone else's selection of the
           data, and it is not one person's, but it has been
16   a team effort in selecting data for use.

17 (Goble Dep. at pp. 96-97).

18   Plaintiffs and Goble essentially argue they were not

19 obligated to consider anything other than what HEDR considered in

20 the way of validating data. Plaintiffs quote from the response

21 of HEDR scientists to a question from the Technical Steering

22 Panel[478] about the failure to use other data sets for HEDR's

23 validation exercise:

24 _____

25   [478] The members of this panel directed the HEDR project
    work. The panel consisted of experts in various technical fields
26 relevant to project work and representatives from the states of
    Washington, Oregon, and Idaho; Native American Tribes; and the
27 public. (HEDR Validation Report at p. iii).
28
    ORDER RE SUMMARY JUDGMENT-   589

1   A considerable body of additional raw data
          does exist for the early years of Hanford
2   operations. However, it is badly fragmented
          in space, time and environmental media. The
3   data 'sets' selected were those that comprise a
          coherent picture of a particular time or place.
4   This is what makes them 'sets,' rather than
          just 'compilations.' Detailed comparisons
5   against the thousands of essentially random
          (in time, space, and medium) measurements would
6   be much beyond the scope of the environmental
          calculations authorized by the TSP (i.e. monthly
7   averages).

8 (HEDR Validation Report, Appendix E at p. E. 17)(Emphasis added).

9   Plaintiffs' counsel contend computer databases pertaining to

10 1948-1951 vegetation data "comprise compilations of historical

11 raw data that had not been evaluated in the course of the

12 protracted quality assurance procedures directed toward the other

13 data used in the HEDR validation exercise . . . and have to this

14 day not been corrected for historical methodological biases by

15 HEDR or any other organization." They say Goble's approach was

16 scientifically reliable in choosing to rely on data sets that had

17 been "quality assured" by scientists not employed by either of

18 the parties for the litigation. They contend the 1948-51 data

19 was not "quality assured" (i.e. "vetted").

20   Vegetation data for 1945-47 are published in Denham, et al.,

21 "Phase I Summaries of Radionuclide Concentration Data for

22 Vegetation, River Water, Drinking Water, and Fish," (1993) (PNWD-

23 2145). Conversion and correction factors for the 1945-47 beta-

24 counting method of I-131 analysis on vegetation are provided in

25 Mart, et al., "Conversion and Correction Factors for Historical

26 Measurements of Iodine-131 in Hanford Area Vegetation," (1993)

27 (PNWD-2133). Vegetation data for 1948-51 are published in Hanf,

28 **ORDER RE SUMMARY JUDGMENT-   590**

1 et al., "Iodine-131 in Vegetation Collected Near the Hanford

2 Site: Concentration and Count Data for 1948-1951," (1993) (PNWD-

3 2177). Conversion and correction factors for the 1948-51 "wet

4 chemistry" method of I-131 analysis on vegetation are provided in

5 Denham, et al., "Conversion and Correction Factors for Historical

6 Measurements of Iodine-131 in Hanford-Area Vegetation 1948-51,"

7 (1993) (PNWD-2176).[479]

8   The fact HEDR figured both conversion and **correction** factors

9 for Hanford vegetation collected in 1945-47 and 1948-51 means

10 this data was "vetted" at least to some extent.[480] The reports

11 listed above were available in 1993, before Goble undertook his

12 calibration. Goble does not deny he had access to databases

13 containing the 1945-47 and 1948-51 vegetation data. (Goble Dep.

14 at pp. 88-90).

15   Plaintiffs' **counsel** specifically identify only one area in

16 which they believe the 1948-51 data was not sufficiently

17 "vetted." This pertains to the "gamma spectrometry" measurement

18 technique which replaced the "wet chemistry" technique in 1957.

19 Plaintiffs' counsel assert that use of the "gamma spectrometry"

20 revealed the "wet chemistry" technique was biased low (it

21 underreported the actual amount of iodine on the vegetation) and

22 _____

[479] HEDR also published an "Overview of Vegetation
23 Monitoring Data, 1952-1983" in 1994 by a J.P Duncan.
   (Defendants' Ex. 26). The vegetation monitoring data after 1951
24 was not used in the HEDR Project because HEDR determined the
   "emissions which affected vegetation were significantly less
25 after 1951." (Duncan 1994 at p. v).

26   [480] Plaintiffs describe "vetted" data as that which has been
   "quality assured" by extensive analysis and **correction.**

27
28 **ORDER RE SUMMARY JUDGMENT-**   **591**

1 this was not adequately taken into account in determining the

2 conversion and correction factors for vegetation data obtained by

3 use of the "wet chemistry" technique.   (See Appendix A to

4 Plaintiffs' Response Brief).[481]

5   As already discussed, a threshold problem is that **Goble**

6 himself never identified this in his reports or at his deposition

7 as an issue which caused him concern about the reliability of the

8 1948-51 vegetation data.   Secondly, whatever the merit of the

9 contention of plaintiffs' **counsel**, it does not necessarily erase

10 the uncertainty of the gross beta counting method which was used

11 for the 1946 vegetation data.   Goble admitted the superiority of

12 the "wet chemistry" technique.   He may have qualified his

13 admission by saying it was necessary for the "wet chemistry"

14 technique to be performed correctly.   However, the fact is **Goble**

15 never pointed to anything specific showing it had not been

16 performed correctly and therefore, that the 1948 to 1951

17

18

19

_____

20   [481] Plaintiffs assert HEDR did not resolve the discrepancy
between the "wet chemistry" technique and the "gamma
21 spectrometry" technique.
      HEDR did not develop any correction or conversion factors
22 for vegetation data after 1951, stating that "[b]eginning in mid-
1951, all parameters had been determined and were being applied
23 to convert net counting rates to activity . . . which provided
accuracy comparable to that of today." Denham, et al.,
24 "Conversion and Correction Factors for Historical Measurements of
Iodine-131 in Hanford-Area Vegetation 1948-1951," (1993) at p. v.
25 According to Denham, et al., "conversion and correction factors
presented in this report were determined by comparing the
26 assumptions used in deriving the 1948-1951 data to processes and
procedures that are standard **today**." (Id. at p. vii)(Emphasis
27 added).

28
ORDER RE SUMMARY JUDGMENT-   592

1 vegetation data could not be used.[482]

2   Defendants point out that the 1949 Green Run data is a part

3 of the 1948-51 vegetation data set and Goble indicated he would

4 use the Green Run data for the purpose of estimating doses for

5 the Green Run.   According to Goble:

> 6   The validation exercises provide data which
>   may be used to make dose estimates independent
> 7   of the use of the HEDRIC modeling suite. To have
>   this capability is useful for two purposes. The
> 8   first is that such estimates provide a further
>   check on model capabilities (either the HEDRIC
> 9   suite, or the calibrated modeling I propose). The
>   second use is that such modeling is likely to be
> 10   preferable in some circumstances. **For instance,**
>   **estimates for someone who received a significant**
> 11   **exposure during the Green run, is more likely to**
>   **be accurate if it is based on measured amounts of**
> 12   **radiation, than if it uses the corrected or un-**
>   **corrected HEDRIC suite.**

13 (Goble 1995 Report at pp. 61-62)(Emphasis added).

14   Essentially, Goble says the Green Run data from December

15 1949, derived from the "wet chemistry" technique, is reliable

16 enough for measuring doses.   There is no indication how Goble

17 might try to distinguish the Green Run data from the other

18 vegetation data of the 1948 to 1951 period, which is derived from

19 the same measurement technique.   Allegations about the

20 unreliability of the 1948-51 data do not originate from Goble.

21 They come from plaintiffs' counsel.   Goble acknowledged that

22 other than the December 1949 Green Run data, he did not do any

23 analysis of the other 1948 to 1951 data.   (Goble Dep. at pp. 90-

24 91).   He also acknowledged that HEDR analyzed the laboratory

25 ─────────────────────

26   [482] At his deposition, Goble alluded in general to there
    being "some problems" early on with the "wet chemistry"
27 technique.   (Goble Dep. at pp. 109-110).

28 **ORDER RE SUMMARY JUDGMENT-   593**

1 methods used to measure vegetation for the 1948 to 1951 time

2 period.   (Id. at p. 91).

3   Goble himself offered different reasons for not using other

4 vegetation data:

> The advantages of the 1946 data compared to the
> later data are basically that the releases were
> larger, so that you have higher values on average,
> and that makes it easier to count. And the other
> advantage is that you don't have to be concerned
> with contamination from atomic testing, which
> began to be a concern for the later testing.
>
> The remaining issues in comparing the data sets,
> comparing 1946 to '49, it is easier to measure,
> I agree, once they had the chemistry going right,
> which they had some problems with, you do have
> cleaner measurements of iodine, and the issues in
> selecting a data base are ones of how many data
> points you have, where they are located, how
> representative are they.   [T]hose are the main issues.

(Goble Dep. at pp. 109-110).

    Asked whether he was testifying that the 1946 releases were

higher than the 1951 releases, Goble said no. He then stated the

1946 releases were higher than those in 1949, but quickly backed

off from that after acknowledging the "large monthly release"

which occurred during the Green Run of December 1949. (Id. at p.

110).

    Goble testified he did not know if there were more

"vegetation measurements" (data points) in 1946 than in 1951.

Asked whether he knew whether the vegetation data from 1951

contained data from more locations than the 1946 data, Goble

responded that he did not recall and that "[i]t exists in a

database we could look up." (Id. at pp. 110-111).

    Asked whether contamination from atomic testing would only

ORDER RE SUMMARY JUDGMENT-   594

1 increase "vegetation concentrations," Goble said "yes." (Goble

2 Dep. at p. 111). This was a concession atomic testing would only

3 increase **non-iodine** concentrations on the vegetation and

4 therefore, that HEDR's estimates of iodine concentrations between

5 1948 and 1951 were more accurate.

6   The emissions from atomic testing were "non-iodine" in

7 nature. In 1946, there was no atomic testing.[483] Hence, there

8 were no non-iodine emissions from atomic testing to further

9 complicate the measurement of iodine via the Geiger-Muller "gross

10 beta" technique. Atomic testing in 1951 meant there was a

11 combination of iodine and non-iodine concentrations on

12 vegetation, making it imperative to distinguish those different

13 types of concentrations. If the "wet chemistry" technique was

14 not able to make that distinction, then the "iodine"

15 concentrations reported for 1951 would have been much higher than

16 they actually were. Plaintiffs' counsel do not contend the "wet

17 chemistry" technique is biased "high" (overreports the amount of

18 iodine). Indeed, they allege it is biased "low" (underreports

19 the amount of iodine).

20   Goble and plaintiffs' counsel offer no compelling reason

21 that atomic testing was any type of factor making the 1946

22 vegetation data more reliable than subsequent vegetation data.

23 It is also noted that in their response brief, plaintiffs'

24 counsel do not cite any of the reasons Goble offered at his

25 deposition for choosing the 1946 data over subsequent data.

26 _____

27   [483] Atmospheric testing.

28 **ORDER RE SUMMARY JUDGMENT-   595**

1   Goble's faith in the reasons offered by him regarding the

2   "advantages" of the 1946 data is best summed up by him: "[A]ll I

3   was doing was listing issues. I wasn't testifying

4   as to exactly how '46 compared with any other particular year."

5   (Goble Dep. at pp. 110-11).

6   Goble was asked why he did not compile all of the Hanford

7   vegetation data for 1944-60 and base his calibration factors on

8   this "complete" set of data. First, he asserted that "other data

9   sets are not as neatly packaged" as the 1946 data set. Secondly,

10   he asserted that compilation of all the vegetation data for 1944-

11   60 would have been an "enormous amount of work" requiring a

12   "HEDR-like effort to make such a data base." Goble stated he did

13   not have "any HEDR computer output for the vegetation

14   predictions," that the only data he had available was "reading

15   off the 1946 sage brush data" and therefore, he "couldn't have

16   made this comparison for other years within any kind of time

17   period that I can offer, I think." (Goble Dep. at pp. 126-27).

18   In other words, Goble contends he could not generate HEDR model

19   predictions for sagebrush contamination for years other than

20   1946. For 1946, those predictions were already provided courtesy

21   of HEDR.

22   First of all, as pointed out above, the vegetation data from

23   other years was summarized and conversion and correction factors

24   provided. It was "packaged" like the 1946 data. Secondly,

25   Goble's deposition testimony itself reveals he did not completely

26   rule out his ability to make a comparison for years other than

27   1946 (between HEDR model predictions and historical data). He

28   **ORDER RE SUMMARY JUDGMENT-   596**

1   acknowledged the HEDR model could be run to produce the sagebrush

2   predictions. (Goble Dep. at p. 127). Goble knew how to extract

3   information from the HEDRIC data tapes. He had those tapes

4   loaded onto a computer from which he generated monthly dose

5   estimates. (_Id_. at pp. 46-52).

6   Lack of time and resources is not a valid excuse for failing

7   to look at other vegetation data. It is irrelevant to the

8   scientific method. Instead of their experts looking at

9   vegetation data for years subsequent to 1946, plaintiffs had the

10   Herrmanns analyze release (source term) estimates for those

11   years. For 1944-47, Goble is willing to accept HEDR's release

12   estimates as modified by his vegetation correction factor based

13   on the 1946 data. For 1948-60, Goble does not restrict himself

14   to a vegetation correction factor based on the 1946 data. He

15   includes a source term correction factor to account for the

16   higher releases estimated by the Herrmanns.

17   By not using any other vegetation data than the Calendar

18   Year 1946 data set, it appears as if Goble's dose estimates

19   **cannot** be validated. First, Goble says he is entitled to rely on

20   the data sets used by HEDR in its validation exercise. However,

21   of those data sets, Goble uses only the Calendar Year 1946 data

22   set. He points out limitations in the other data sets- April 13,

23   1946 footprint data set, December 1949 Green Run data set,

24   September 1963 PUREX Release data- which he contends make them

25   unsuitable for his calibration purposes. While HEDR contends the

26   data sets present a **"coherent picture of a particular time or**

27   **place,"** plaintiffs and apparently Goble disagree with that

28
     ORDER RE SUMMARY JUDGMENT-   597

conclusion. For instance, HEDR seems to say the 1949 Green Run

data presents a coherent picture of iodine concentrations on

vegetation in the late 40s' and early 50s'. Goble seems to say

it only presents a coherent picture of iodine concentrations on

vegetation for the month of December 1949.

Goble does not specifically validate his dose estimates

against any of data selected by HEDR. Instead, he asserts he

compared the "general consistency or inconsistency" of his

predictions with the data selected by HEDR. Plaintiffs' counsel

attempt to perform a validation on Goble's behalf with regard to

the Footprint data, the Green Run data, and the PUREX data,

acknowledging the Calendar Year 1946 data set cannot be used to

"validate" Goble's dose estimates (model predictions). However,

they qualify their validation attempt by saying it is necessary

for them to assume it is **proper** to derive long-term average

measurements from HEDR's data sets (other than the Calendar Year

1946 data set). Of course, plaintiffs and apparently Goble

contend those data sets cannot **properly** be used for such

purposes. Although the 1948-51 data set would seem to meet their

criteria as a "time sequence" or "long-term" data set, plaintiffs

and apparently Goble assert it cannot be used for comparison

purposes.[484]

---

[484] As defendants point out, plaintiffs' demand for "long-
term data sets" is interesting in that Goble's calibration
exercise was motivated by HEDR's significant underreporting of
iodine concentrations for just three months- December, January
and February 1946. For the other months of 1946, HEDR reported
being within a factor of three of the historical measurements,
meeting HEDR's validation objective.

ORDER RE SUMMARY JUDGMENT-   598

1   In the final analysis, the court is left with the distinct

2 impression that there is an attempt to dodge a comparison of

3 Goble's dose estimates with any historical data. If Goble's dose

4 estimation method cannot be validated, it cannot be

5 scientifically reliable.

6

7   **(6) <u>Daubert</u> Criteria**

8   **(a)   Pre-Litigation Research**

9   The two most important criteria for determining whether an

10 opinion is scientifically reliable are whether the opinion grows

11 out of pre-litigation research and whether the research has been

12 subjected to peer review.

13   Defendants contend Goble's opinions regarding environmental

14 radiation dose reconstruction models and historical radionuclide

15 measurements do not grow naturally or directly out of any

16 research he has conducted independent of this litigation. They

17 claim his work is a "litigation construct."

18   Defendants note the following from Goble's deposition

19 testimony: 1) he has never written a paper or report providing

20 the principles to follow in comparing an environmental dose

21 reconstruction model to measured data (Goble Dep. at p. 74); 2)

22 he has never published an article regarding methods for testing

23 dose reconstruction models (<u>Id</u>. at p. 225); 3) he has not done

24 a historical study of developments in vegetation monitoring (<u>Id</u>.

25 at 138); 4) he has not done any historical study of radiation

26 monitoring practices in the 1940s and 1950s (<u>Id</u>. at pp. 247-48);

27 4) he has not conducted a program for monitoring iodine or any
28

ORDER RE SUMMARY JUDGMENT-   599

1 type of fission product in the environment (Id. at p. 232); 5) he

2 has not conducted laboratory analysis for the measurement of

3 radionuclides on vegetation as part of an environmental

4 monitoring program (Id. at p. 137); 6) he has not published an

5 article regarding methods for monitoring any type of

6 radionuclides in the environment (Id. at pp. 224-25); 7) he has

7 not published an article in a peer-reviewed journal regarding an

8 environmental dose reconstruction he has performed (Id. at pp.

9 229-30); 8) he has never been a member of the National Council

10 for Radiation Protection and Measurements (Id. at p. 222); and 9)

11 he is not a certified health physicist (Id. at p. 223).

12   Based on the foregoing, defendants assert Goble is not

13 qualified to "perform radiation monitoring or to evaluate

14 radiation monitoring."[485] It appears defendants do not take

15 issue so much with Goble's qualifications **in general** to

16 "calibrate" a dose reconstruction model.[486] The problem,

17 however, is the significance of vegetation monitoring techniques

18 to the particular "calibration" undertaken in this case. Goble's

19 resume and his deposition testimony do not manifest an expert's

20 familiarity with the type of I-131 measurement techniques used at

21 _____

       [485] Defendants never assert lack of qualifications under FRE
22 702 as a specific and separate legal basis for excluding Goble's
    opinion. It comes up only in connection with whether Goble's
23 work is the result of pre-litigation research, which is one of
    the criteria for determining scientific reliability.

24
       [486] A review of Goble's resume indicates he has experience
25 in general with atmospheric transport and deposition, as well as
    use of models for assessing the consequences of large scale
26 releases of radioactive materials to draw inferences regarding
    emergency planning for nuclear power plant accidents. (Goble
27 1995 Report at p. 98).

28 **ORDER RE SUMMARY JUDGMENT-   600**

1   Hanford over the years. In his reports, Goble did not discuss

2   differences in vegetation monitoring techniques and he did not

3   account for those differences in his "calibration." As

4   discussed, **plaintiffs' counsel** are responsible for the discussion

5   of purported biases in the "wet chemistry" method versus the

6   subsequent gamma spectrometry method.

7   Plaintiffs contend Goble's work (calibration) is a "natural

8   extension" of HEDR which HEDR never got around to following

9   completion of its validation exercise. Plaintiffs say HEDR never

10 got around to it because of limitations of time and resources.

11 Plaintiffs assert that Goble's work, which they describe as

12 "correcting the HEDR model to result in more accurate estimated

13 doses for 'real' individuals," was "required to elevate HEDR to

14 better science."

15   The specific opinion at issue here is Goble's opinion that

16 HEDR model predictions systematically underestimate the measured

17 concentration of iodine on vegetation. This specific opinion was

18 not derived from pre-litigation research. Prior to this lawsuit,

19 there is no indication Goble was engaged in any analysis of HEDR

20 model predictions. This, in itself, does not mean Goble's

21 opinion is the product of an unscientific methodology. However,

22 as the U.S. Supreme Court recognized, it is relevant to the

23 question of scientific reliability.

24

25   **(b) Peer Review**

26   Goble acknowledges none of the reports he prepared for this

27 litigation have been published in a peer-reviewed scientific

28 **ORDER RE SUMMARY JUDGMENT-   601**

1 journal; he has not submitted his reports to any public group for

2 review or comment; and he has not provided them to any experts or

3 group of experts (outside the plaintiffs' experts) for review or

4 comment.   (Goble Dep. at pp. 173-74).   He likewise acknowledges

5 he had not provided any of his criticisms to the TSP or to the

6 States of Washington, Oregon and Idaho who are developing the

7 Individual Dose Assessment Project (IDAP).   (Id. at p. 166).

8   The plaintiffs appear not to dispute most of this, although

9 they claim Goble has provided his criticisms to the Washington

10 Department of Health and Radiation Protection and to Dr. John

11 Till, **former** Chair of the TSP.[487] Plaintiffs assert that too

12 much emphasis is placed upon formal peer review and publication

13 of work in peer-reviewed journals.   Rather, they contend the

14 question is whether Goble's work meets the "qualities for

15 publishability."   According to plaintiffs, the fact Goble's work

16 has not been formally peer-reviewed is of no consequence since

17 "his method is the HEDR method that did undergo extensive peer

18 review by several organizations."

19   Goble's method is not precisely the same as HEDR's method.

20 Goble's "calibration" analysis has not been subjected to formal

21 peer review.   The Ninth Circuit has made it clear that peer

22 review is one of the two principal ways for insuring scientific

23 evidence meets the "reliability" prong of Daubert.   According to

24 the circuit, if the research is accepted for publication in a

25 reputable scientific journal after being subjected to peer

26 _____

    [487] There is no indication what, if any, feedback Goble

27 received from these sources.

28 **ORDER RE SUMMARY JUDGMENT-   602**

1   review, it is a "significant" indication the research is taken

2   seriously by other scientists. Daubert II, 43 F.3d at 1318-19.

3   Plaintiffs note Goble was invited to attend the Center for

4   Disease Control/IDA (Individual Dose Assessment) Workshop in

5   August 1997. They say he was invited to present his critique of

6   the HEDR model and discuss his methodology as an "expert."

7   According to plaintiffs, Goble informed Bruce Napier, HEDR Chief

8   Scientist, of errors in the HEDR model with which Napier agreed.

9   As discussed above, there was acknowledgement on the part of HEDR

10 of the need for a wet/dry ratio in making model predictions.

11 Plaintiffs assert "the major points of [Goble's] calibration have

12 been peer-reviewed through a collegial process . . . ."

13   The fact remains, however, that there has not been a formal

14 peer review of Goble's calibration analysis. Furthermore, the

15 wet/dry ratio is but one part of the calibration. This one

16 correction does not make Goble's analysis scientifically reliable

17 as a whole. Furthermore, it appears **Cochran** was the one who

18 discovered the error in the wet/dry ratio. The courts finds

19 nothing in the record showing Goble was responsible for

20 discovering any of the HEDR errors identified above, including

21 Napier's concession that I-131 concentrations measured during the

22 Green Run needs to be increased by a factor of 2.5.

23

24   **(c)   General Acceptance**

25   Plaintiffs contend Goble's work is "generally accepted"

26 within the scientific community because it is essentially the

27 HEDR methodology. They observe that "calibration" of dose

28 **ORDER RE SUMMARY JUDGMENT-   603**

1 reconstruction models is "well accepted scientific methodology."

2   Plaintiffs assert the court's expert, Dr. Thomas H. Pigford,

3 agreed with most of their criticisms of HEDR, "particularly

4 regarding use of vegetation data for years other than 1946 for

5 validation." They cite the following passage from Dr. Pigford's

6 1994 report ("Assessment of Radiation Dose Estimates Made by

7 Hanford Environmental Dose Reconstruction Project"):

8   It should be recognized that the validation
           results . . . for the 1963 [PUREX] release and
9   for releases in 1946, 1948, and in the 1980s . . .
           do not necessarily indicate the accuracy of these
10   models if applied to the routine operational
           releases that have occurred at Hanford.

11 (Pigford 1994 at p. 17).

12   They also note Dr. Pigford's conclusion that:

13   Several technical issues have been identified in
           this report that indicate possible increases in
14   uncertainty of HEDR's estimates of iodine-131
           releases. Some could result in a larger range
15   of uncertainty in amount released, which could
           translate into increases in calculated doses and
16   in uncertainty in those doses.

17 (Id. at p. 30).

18   Although Dr. Pigford agreed there were limitations

19 potentially affecting HEDR's accuracy in predicting doses, he

20 obviously never reviewed Dr. Goble's "calibration" analysis and

21 placed his seal of approval upon it. Plaintiffs cannot say Dr.

22 Pigford would have agreed it was proper to isolate the Calendar

23 Year 1946 vegetation data set for the purpose of "calibrating"

24 HEDR's doses from 1944 all the way through 1960.

25   The same holds true with regard to Dr. Ruttenber. Although

26 Ruttenber contends he never suggested Goble's work was

27
28 **ORDER RE SUMMARY JUDGMENT-   604**

1   litigation-driven, he does not endorse Goble's work. Ruttenber

2   makes clear that he had "not studied the dose reconstruction work

3   of Dr. Goble enough to form an opinion of any sort, and probably

4   never would, as this type of work is outside my areas of

5   expertise." (Ruttenber Declaration at p. 2, Ex. 8 to Plaintiffs'

6   Appendix I re Iodine Claims).

7   Plaintiffs contend defendants have "failed to cite a single

8   expert report refuting the basic scientific methodology employed

9   by Dr. Goble or his conclusion that actual doses were higher than

10 HEDR predicted." Plaintiffs say that "[b]ecause no expert could

11 possibly quarrel with Dr. Goble's methodology," the defendants

12 have found it necessary that their lawyers do the testifying.

13   It is true that in conjunction with their original

14 submission, defendants did not include any expert critique of

15 Goble's work. However, that is not proof Goble's work

16 constitutes "generally accepted" scientific methodology. The

17 deficiencies in Goble's work are manifest from Goble's own

18 deposition testimony, cited above, and from the various HEDR

19 documents referred to above.

20   The HEDR documents make clear there was vast improvement in

21 I-131 measurement techniques between 1946 and 1948. Goble had no

22 specific basis for refuting that conclusion and indeed, admitted

23 the improvement in measurement techniques. Goble acknowledged he

24 did not incorporate the uncertainty of the 1946 measurement

25 technique into his "calibration" analysis. He simply did not

26 discuss the difference in measurement techniques. Goble

27 acknowledged that "calibrated" results need to be validated

28 **ORDER RE SUMMARY JUDGMENT-   605**

against measurement data, but he undertook no specific

validation. Indeed, as discussed above, it is as if Goble's

analysis makes it so there is no data against which his

calibration can be validated. At his deposition, Goble

acknowledged he would use HEDR's sagebrush-to-pasture adjustment.

He offered no excuse for his failure to consider that adjustment

in his "calibrated" results. Finally, the court must note that

plaintiffs' counsel endeavor to fill the gaps left by Goble-

i.e. discussion re limitations of "wet chemistry" method;

validation of Goble's results specifically as against 1946

footprint data; 1949 Green Run data; and 1963 PUREX data.

While "calibration" of dose reconstruction models is an

accepted scientific principle, there is no indication the

particular "calibration" undertaken by Goble in this case has

been "generally accepted" within the scientific community.[488]

(d) Testing of the Method

Goble did not undertake a specific validation of **his** model

predictions with HEDR's validation data sets, or any other data.

---

[488] Plaintiffs' expert Dr. Ghiselin offers very general
observations about what constitutes proper scientific method. He
does not address the specific issues concerning the
methodological soundness of Goble's calibration analysis and
indeed, by his own admission is not qualified to do so:

> I do not possess the specialized expertise that
> would qualify me to carry out the kind of study I
> have been asked to evaluate.

(Ghiselin Affidavit at p. 3).

ORDER RE SUMMARY JUDGMENT-   606

1 Rather, he asserted his results were "generally consistent" with

2 HEDR's validation data sets, which plaintiffs label "non-

3 representative" on the basis that they are not "long-term average

4 data."   Plaintiffs' **counsel** undertook the task of attempting to

5 specifically validate Goble's work against HEDR's validation data

6 sets.

7   While **Goble's** method can be tested, **he** did not test it.

8

9   **(e)   Potential Rate of Error**

10   Goble admits his calibration analysis did not specifically

11 quantify and incorporate the uncertainty in the 1946 vegetation

12 measurements due to use of the gross beta counting method.

13 Therefore, he did not account for a known potential rate of

14 error.

15

16   **c.   Conclusion**

17   The court will grant defendants' motion in limine to exclude

18 Dr. Goble's reports.   Dr. Goble will not be allowed to testify at

19 trial.

20   The primary reasons for this are his failure to adequately

21 and specifically consider the limitations of the 1946 vegetation

22 data, his failure to consider any other vegetation data, and

23 reasoning by him which appears to ultimately make his method

24 "validation-proof."[489]   Furthermore, it is clear Goble's reports

25 _____

26 [489]   Goble's failure to use a sage-to-pasture adjustment is
    not impressive, particularly since at his deposition he did not
    acknowledge an oversight on his part, but just matter of factly

27 stated he would use HEDR's sage-to-pasture adjustment.   Although

28 **ORDER RE SUMMARY JUDGMENT-   607**

1 do not meet the circumstantial guarantees of reliability found in

2 the Daubert criteria.

3   While plaintiffs contend "selectivity of data" is an issue

4 which goes to the weight (sufficiency) of Dr. Goble's opinion,

5 and not its admissibility, that is not true in this particular

6 situation. This court, in this very litigation, has expressed

7 its concern about experts who are selective in their choice of

8 supporting data and who focus only on fragments of data which

9 lend credence to their theories. In re Hanford Nuclear

10 Reservation Litigation, 894 F.Supp. 1436, 1450 (E.D. Wash. 1995)

11 (excluding opinion of plaintiffs' expert, Dr. Thomas L. Welsh).

12   While this court cannot and does not reach any conclusion as

13 to whether HEDR's underprediction of doses versus 1946 vegetation

14 measurements is in fact due to limitations of the gross beta

15 counting method, or due to deficiencies in the model itself, the

16 record makes it abundantly clear Goble should have at least

17 considered the limitations of the gross beta counting method and

18 incorporated the uncertainty thereof in his calibration. He also

19 should have made an attempt to specifically validate **his** model

20 predictions against other vegetation data, including data

21 specifically considered by HEDR in its validation exercise, as

22 well as data not considered by HEDR.

23   Exclusion of Goble's opinion does **not** constitute an

24 endorsement by this court of HEDR's predictions. As this

25 _____

26 the court does not conclude this was a deliberate effort to
   inflate his doses, it certainly does not bolster confidence in
27 the rest of his work.

28 **ORDER RE SUMMARY JUDGMENT-   608**

1 discussion has revealed, HEDR has its share of flaws. This court

2 is not concluding that HEDR's predictions are "validated" and

3 Goble's predictions are not "validated." Rather, it concludes

4 only that Goble's failure to "validate" his results and consider

5 the uncertainty of the 1946 measurements makes **his** methodology

6 scientifically unsound. That is a question of "admissibility" as

7 opposed to the "weight" to be given different conclusions.

8

9 **2. Thomas Cochran**

10   Thomas Cochran, Ph.D., is a Senior Scientist with the

11 Natural Resources Defense Council, Inc. He is the Director of

12 the NRDC's Nuclear Program. He authored several reports for this

13 litigation, including: 1) a March 19, 1996 "revised" report

14 entitled "Errors in the Source Term of the Hanford Environmental

15 Dose Reconstruction Project;" and 2) a March 28, 1996 "revised"

16 report entitled "Calibration of the Hanford Environmental Dose

17 Reconstruction Using Vegetation Data."

18   In their motion in limine directed at Dr. Cochran, the

19 defendants do not, at least explicitly, rely on a <u>Daubert</u>

20 analysis. However, one basis for defendants' motion is that

21 Cochran's work "is so preliminary and incomplete that plaintiffs

22 cannot fairly rely on it to satisfy their generic causation

23 burden of proof." Defendants argue Cochran's work cannot be used

24 to calculate individual iodine doses and therefore, does nothing

25 to assist the trier of fact in answering the inquiry whether an

26 individual received a dose in excess of the doubling dose. This

27 debate appears to involve the "fit" or "relevancy" prong of

28 **ORDER RE SUMMARY JUDGMENT-   609**

1 _Daubert_.

2   Defendants also contend Cochran's work cannot be reconciled

3 with the work of Dr. Goble. They argue plaintiffs should be

4 restricted to relying upon Goble for their dose estimation

5 method. The court is excluding Dr. Goble from testifying about

6 his dose estimation method.

7

8   **a.   Summary of Cochran's Methodology**

9   In his March 19, 1996 "revised" report dealing with source

10 term, Cochran concludes the output data from HEDR's source term

11 (STRM) model for the period 1944-47 is unreliable for individual

12 dose calculations "and consequently the radioactivity measured on

13 and in vegetation during the same period represents a more

14 reliable starting point for estimating individual doses." (March

15 19, 1996 Report at p. 9). Hence, similar to what Dr. Goble does,

16 Cochran proposes to "calibrate" HEDR model outputs using the

17 Calendar Year 1946 vegetation data ("3500 samples reported for

18 1946"). (March 28, 1996 Report at p. 1).[490]

19   In his analysis, Dr. Goble relies upon Cochran in several

20 respects, including the use of a wet/dry ratio to correct HEDR

21 model calculations. While HEDR model calculations were performed

22 in terms of dry weight (i.e. assumed all the moisture had been

23 removed from the sagebrush samples), HEDR's Validation Report

24 _____

25   [490] According to defendants, what Cochran does is "estimate
doses for a narrowly circumscribed area directly from the 1946
26 vegetation data, rather than running the data through the complex
HEDR model (or any similar model) as Goble purports to have
27 done."
28
ORDER RE SUMMARY JUDGMENT-   610

1 "erroneously failed to include a conversion from dry weight to

2 wet weight concentrations before comparing the output to the

3 measurement data." (March 28, 1996 Report at p. 3). HEDR

4 acknowledges this error. Cochran proposes a 2.25 wet/dry ratio

5 which plaintiffs concede mistakenly includes "bud sagebrush,"

6 requiring the ratio be lowered.

7 Incorporating this "wet/dry" correction, as well as several

8 other "corrections," Cochran concludes the HEDR model

9 underpredicts the concentration of I-131 in sagebrush "by a

10 factor that varies from about 7 to 10 in summer to about 13 to 33

11 in winter." (Id. at p. 5). The vegetation correction factor

12 used by Goble falls within those ranges: 8.7 for the summer

13 months and 26.3 for the winter months.

14 Cochran goes through each of HEDR's validation data sets in

15 an effort to see how his vegetation correction factor range

16 compares to those data. For example, with regard to the April

17 13, 1946 "footprint" data, Cochran concludes HEDR underpredicts

18 I-131 vegetation concentration by a factor of about nine which he

19 says "reinforced" his conclusion that "the HEDR models

20 underpredicted I-131 concentration in vegetation by about a

21 factor of 7 to 10 in the early years during the summer months."

22 (Id. at p. 7).

23 With regard to the 1949 Green Run data, Cochran opines that

24 HEDR underpredicts I-131 concentration in vegetation by a factor

25 of 3.15. Cochran points out several factors in favor of giving

26 more weight to the Green Run data and several factors for giving

27 it less weight. The factors favoring more weight include: 1)

28 **ORDER RE SUMMARY JUDGMENT-   611**

1 the Green Run was a controlled experiment; 2) there were more

2 geographical cells involved[491]; and 3) **measuring equipment and**

3 **techniques had improved.** Cochran cites the following reasons for

4 giving less weight to the Green Run data: 1) the experiment was

5 not contemporaneous with the period of greatest releases from the

6 chemical separations plants; 2) the pattern represented by the

7 location of the vegetation measurements was not as uniform; 3)

8 the counting laboratory was contaminated; 4) there was

9 significant uncertainty in the conversion factor that should be

10 applied to the raw CPM (counts per minute) data to obtain I-131

11 concentrations, and the effective weathering half-life assumed by

12 the HEDR model was not in evidence; and 4) HEDR predictions

13 appeared less accurate with increasing time. Cochran concludes

14 that considering all of these factors, the Green Run data

15 provides "no basis for altering the conclusion that HEDR models

16 underpredict I-131 concentrations in vegetation by at least a

17 factor of seven or more in the early years." (Id. at p. 8).

18   With regard to the September 1963 PUREX data, Cochran finds

19 that "[s]ince only one [geographical cell] was sampled[492], these

20 limited 1963 data provide no basis for altering the conclusion

21 that the HEDR models underpredict vegetation concentrations by at

22 least a factor of seven and more in the early years." (Id. at p.

23 11).

24   Although Goble relies on Cochran in certain respects, he

25 _____

26 [491] The Calendar Year 1946 vegetation data was obtained from five geographical cells: 440, 442, 443, 467 and 469.

27 [492] Geographical cell number 468.

28 **ORDER RE SUMMARY JUDGMENT-   612**

1 clearly goes much further than Cochran with respect to dose

2 estimation analysis. Cochran's analysis is much more

3 "preliminary" in nature. Cochran states that his correction

4 factors (i.e. wet/dry ratio for concentration of I-131 in

5 sagebrush; sagebrush to pasture ratio; pasture (feed) to milk

6 ratio) apply "to the component of the 1946 thyroid dose delivered

7 through the grass-cow-milk food chain, where the individual is

8 getting his/her milk from a family (backyard) cow, and the **dose**

9 **is only for 1946 in the cities identified above** [North Richland,

10 South Richland, Kennewick/Pasco and Benton City]."[493] (March

11 28, 1996 Report at p. 20)(Emphasis added). Table 12 of Cochran's

12 March 28, 1996 Report at p. 41 provides "Thyroid Doses[s] From

13 1946 Intake of Milk From Cows on Fresh Pasture Grass" for: 1) a

14 three month old infant; 2) a four year old male; and 3) an adult

15 male. However, Cochran adds that:

16   . . . the process by which these results
       were computed **can be extended to other years,**

17   **locations, feeding regimes, etc. While the**
       alternative exposure pathways are too numerous

18   **to explain how this would be done in each case,**
       I will sketch out some of the more important

19   **considerations.**

20 (Id. at p. 20)(Emphasis added).

21   Among those "considerations" is correction of source term

22 estimates. According to Cochran, the second page of his Table 12

23 (March 28, 1996 Report at p. 42) shows "how one can further

24 ───────────────────

25   [493] These are the areas from which the Calendar Year 1946
   vegetation data set was collected. North Richland is Cell 469.

26 South Richland is Cell 442. Kennewick/Pasco is Cell 443. Benton
   City is Cells 440/467. (See Table 12 of Cochran's March 28, 1996

27 Report at p. 41).

28 **ORDER RE SUMMARY JUDGMENT-   613**

1 **(partially)** correct the HEDR dose estimates for other years for

2 the same cities." (<u>Id</u>. at p. 21)(Emphasis added). In addition

3 to 1946, Cochran provides thyroid doses for the "Fresh Pasture-

4 Family Cow-Milk Pathway" for 1944-45 and 1947 through 1953 for a

5 three month old infant and an adult male. (<u>Id</u>. at p. 42).

6   Cochran is unwilling to commit himself to doses for any

7 years other than 1946. Cochran derives those 1946 doses from

8 what he refers to as HEDR's "uncorrected" annual release

9 estimates, and states he will "stick with [those] annual values

10 until the HEDR source term model is **corrected**." (<u>Id</u>. at p.

11 21)(Emphasis added). Cochran says he has identified several

12 errors and inconsistencies in the I-131 source term for the

13 period 1944-49. (<u>Id</u>. at p. 20). For the period 1950-1972,

14 Cochran states he would increase the HEDR source terms to correct

15 for the bias introduced as a result of HEDR's use of monthly

16 average cooling times. He said he "would" also correct for other

17 source term errors identified by other experts, notably the

18 Herrmanns. (<u>Id</u>. at p. 21). At the time of his deposition,

19 Cochran was still unwilling to commit himself to a specific

20 source term release factor for the purpose of computing doses for

21 years other than 1946. (Cochran Dep. at p. 249-256).

22   In contrast, Goble is content to use HEDR's source term

23 estimates for 1944-47. Furthermore, for the period 1948-60,

24 Goble commits himself to the Herrmanns' source term release

25 estimates (source term correction factor of 20). As such, unlike

26 Cochran, it is possible to extrapolate Goble's analysis to years

27 other than 1946 and Goble is willing to commit to doses for 1944-

28 **ORDER RE SUMMARY JUDGMENT-   614**

1   45, 1947, and 1948-60.

2   Goble also specifically considers "alternative exposure

3   [ingestion] pathways" besides the backyard cow pathway, including

4   milk (backyard cow and **processed** milk), beef, eggs, fruit, grain,

5   leafy vegetables and other vegetables. The only milk pathway

6   Cochran analyzes is the "backyard cow" pathway." While he

7   acknowledges that "[t]he growth rate constants and the density of

8   the biomass will be different for vegetables other than sagebrush

9   and pasture grass," Cochran states "[t]hese differences **can** be

10 taken into account **when** calculating thyroid dose components due

11 to other pathways." (March 28, 1996 Report at p. 21)(Emphasis

12 added). Cochran also does not indicate how doses would be

13 calculated for the inhalation pathway, other than that it would

14 not include a "seasonal factor" which he includes in calculating

15 doses for the backyard cow pathway. (Id.)

16   Goble employs a "distance correction factor" for deriving

17 dose estimates for other locations more distant than North

18 Richland, South Richland, Kennewick/Pasco and Benton City.

19 Cochran alludes to Goble's "distance correction factor" and the

20 need to "reduce the [vegetation] correction factor to account for

21 depletion of the plume." (Cochran March 28, 1996 Report at p.

22 21). However, Cochran never commits himself to using Goble's

23 distance correction factor. (Cochran Dep. at p. 258).

24

25   **b. Reliability**

26   Defendants do not specifically assert this prong of <u>Daubert</u>

27 as a reason for excluding Cochran's "calibration." However,

28 **ORDER RE SUMMARY JUDGMENT-   615**

because it is scientifically unreliable for Goble to rely on the 1946 vegetation data for "calibration" purposes due to his failure to quantify and incorporate the uncertainty of the "gross beta counting" measurement technique, it follows that Cochran's "calibration" is likewise scientifically unreliable.

In his March 28, 1996 Report, Cochran states one of the reasons for giving more weight to the Green Run data is because **"measuring equipment and techniques had improved."** Interestingly, however, Cochran testified at his deposition that he did not have a "specific technology improvement in mind" and furthermore, he could not recall if he read that Hanford had switched from a "gross beta counting" method to a "wet chemistry" method in 1948. Rather, he more or less assumed there was an improvement in the measurement technique. (Cochran Dep. at pp. 260-61). Cochran did not attempt to quantify the uncertainty of the "gross beta counting" method. He did not analyze the impact of different measurement techniques. Furthermore, Cochran stated he had not reviewed later vegetation data, in particular 1951 data, but acknowledged it would be a "useful exercise" which had the potential for modifying his "views." (Cochran Dep. at pp. 278-79). Cochran did not compare his predictions with any data independent of the 1946 vegetation data. (Id. at p. 273).

Exclusion of Cochran's "calibration" analysis is warranted based on the "reliability" prong alone.

c. Conclusion

The court will grant defendants' motion in limine re Thomas

ORDER RE SUMMARY JUDGMENT-   616

Cochran on the basis that his methodology is scientifically

unreliable because of his failure to adequately consider the

limitations of the Calendar Year 1946 data, in particular the

measurement technique used for collecting that data. This is the

basis on which Goble's analysis is being excluded. Exclusion of

Goble's analysis necessitates exclusion of Cochran's

analysis.[494]

3. Alexandre Klementiev

In addition to his retention by plaintiffs for the purpose

of performing a plutonium source term analysis, Dr. Klementiev

was retained to perform a source term analysis of iodine (I-131)

emissions. In November 1995, he prepared a report entitled

"Estimation of the Iodine-131 Releases to the Atmosphere from the

Hanford Site (1944-60)." Defendants move to exclude Klementiev

from testifying about his iodine source term analysis, contending

it is scientifically unreliable and that he is not qualified to

offer an expert opinion regarding this subject.

a. Overview of Plutonium Production Process

HEDR and Dr. Klementiev considered three factors in

determining the amount of I-131 emissions: 1) I-131 Creation; 2)

I-131 Decay; and 3) I-131 Release Factor.

---

[494] The court should make clear that it contemplated dose
reconstruction models would be complete at the end of Phase II so
as to expedite the calculation of individual doses during Phase
III. Plaintiffs recognized this as well. See January 25, 1996
Third Order Re: Case Management Discovery Plan at pp. 4-5 (Ct.
Rec. 632).

ORDER RE SUMMARY JUDGMENT-   617

1   I-131 is a by-product of the plutonium production

2   process.[495]   At Hanford, plutonium was created by placing

3   uranium metal rods inside a reactor and bombarding them with

4   neutrons.   This caused some of the uranium atoms to be

5   transformed into plutonium while other uranium atoms split apart

6   to form I-131 (radioiodine), a "fission product."   The amount of

7   I-131 created within the uranium metal rods or "fuel slugs" is a

8   function of reactor power levels, duration of slug exposure, and

9   slug location within the reactor.

10   After the desired amount of plutonium formed inside the

11   slugs, the slugs were removed from the reactor and stored for a

12   specified period of time known as the "cooling time."   During

13   this time, the radioactivity of the slugs decreased through

14   radioactive decay.   "I-131 Decay" refers to the amount of I-131

15   that decayed out of the uranium fuel slugs while they cooled

16   between their time in the reactor and their being dissolved in

17   nitric acid at the start of the separations process.

18   To recover the plutonium, the fuel slugs were brought to a

19   chemical separations plant (T-Plant, B-Plant, REDOX or PUREX) and

20   dissolved in nitric acid.   The resulting solution then underwent

21   chemical extraction and purification processes.   During this

22   dissolving process, some of the I-131 inside the slug was

23   released in gas form to the atmosphere via the separations plant

24   stacks.   "I-131 Release Factor" refers to the fraction of I-131

25   in the cooled slugs released from the separations plants during

26   _____

[495]   As are some of the other non-iodine elements discussed
27   in passing, including cerium, cesium, ruthenium and strontium.

28   **ORDER RE SUMMARY JUDGMENT-   618**

1 the separations process.

2

3   **b. 1944-49 Source Term Analysis**

4   **(1) HEDR Analysis**

5   For the 1944 to 1949 time period, HEDR estimates 695,971

6 curies (Ci) of I-131 were released. HEDR reviewed historical

7 operational records to reconstruct the handling of each batch of

8 uranium fuel slugs irradiated in the reactors and subsequently

9 dissolved. HEDR reviewed the historical records on reactor

10 operations and uranium slug cooling times to determine the amount

11 of iodine present in each batch of slugs at the time of its

12 discharge from the reactor and when the batch was dissolved in

13 the nitric acid. Based on the amount of I-131 available for

14 release at the time the slugs were dissolved, HEDR applied a

15 release factor to determine the amount of I-131 emitted from the

16 stacks of the separations plants. See generally, C.M. Heeb,

17 "Iodine Releases from the Hanford Site, 1944-1947: Vol. 1 Text,"

18 (PNWD-2033)(March 1993) (hereinafter, "Heeb 1993 Vol. 1"); C.M.

19 Heeb, "Iodine Releases from the Hanford Site, 1944-1947: Vol. 2

20 Data," (PNWD-2033)(March 1993) (hereinafter, "Heeb 1993 Vol. 2");

21 and C.M. Heeb, "Radionuclide Releases to the Atmosphere from

22 Hanford Operations, 1944-1972," (PNWD-2222)(May 1994)

23 (hereinafter, "Heeb 1994").[496]

24   This information was plugged into the HEDR computer

25 programs, specifically the Reactor Model (RM) and the Source Term

26 _____

27   [496] Defendants' Exs. 47, 48 and 49.

28
ORDER RE SUMMARY JUDGMENT-   619

1   (STRM) programs. The "RM" is designed to calculate the amount of

2   I-131 being created in the reactors. The "STRM" measures the

3   radionuclide release rate, in this case, I-131 released from the

4   dissolving (dissolution) process. Id. The result is HEDR's

5   estimate that 695,971 Ci of I-131 were released between 1944 and

6   1949. (Heeb 1994 at Table S.1, p. vii).

7

8   **(2) Klementiev Analysis**

9   Klementiev developed two separate models to estimate iodine

10   emissions for 1944-49. His "Alternative Reactor Model" or "ARM"

11   was designed to estimate the amount of I-131 present in uranium

12   fuel slugs when discharged from the reactor. His "Model for the

13   Estimation of Releases of Iodine" or "MERI+" was designed to

14   estimate the amount of I-131 released from the separations

15   plants, based on the output of ARM. ARM and MERI+ ostensibly are

16   alternatives to HEDR's "RM" and "STRM" codes.

17   Klementiev acknowledges he did not use the output of his ARM

18   model, although in his report he stated the model "was developed

19   to produce **independent** estimates of I-131 concentrations in the

20   fuel discharged from Hanford reactors in the early years of

21   production." (Klementiev 1995 Report at p. 7)(Emphasis added).

22   According to Klementiev, he ran his ARM model for the periods

23   November 1944 to March 1946 (B Reactor), January 1945 to March

24   1946 (D Reactor) and July 1945 to March 1946 (F Reactor). He

25   states he found his "results were essentially the same as the

26   results obtained from the HEDR reactor model [RM]." In order to

27   save time, he began to use the output of HEDR's "RM."

28   **ORDER RE SUMMARY JUDGMENT-   620**

1 (Klementiev 1997 Affidavit at pp. 5-6).[497] Klementiev used

2 HEDR's "RM" output for all the months of operation between 1944

3 and 1949.

4   Klementiev acknowledges the "mathematical description" of

5 his MERI+ model is "similar" to the corresponding model offered

6 by HEDR (STRM), such that both MERI+ and STRM "give the same

7 result when the inputs are the same." (Klementiev 1997 Affidavit

8 at p. 4). However, he changed the input of MERI+ "in accordance

9 with the **suggestion that FIFO was slightly violated** . . . ."

10 (Id.)(Emphasis added).

11   "FIFO" refers to the "first-in, first-out" rule which is

12 that uranium slugs placed first in the reactor will be the "first

13 out" of the reactor and the first to be dissolved in nitric acid.

14 This is apparently the protocol which Hanford contractors tried

15 to follow in order to keep I-131 releases at a "safe" level. The

16 "oldest" fuel slugs were those which would have "cooled" the

17 longest and therefore, experienced the most radioactive decay

18 prior to being dissolved in the nitric acid and give off the

19 least I-131 gas.[498]

20   Klementiev found the output of his MERI+ model increased by

21 32% if on average about 6% of the fuel was "transposed" (i.e.

22 newer or "greener" fuel dissolved before the oldest fuel), and by

23 64% if on average one bucket (of slugs) out of eight was

24 "transposed." This results in what Klementiev calls "adjustment"

25 _____

[497] Foulds Ex. 70.

26

[498] Klementiev also refers to FIFO as the "Oldest Fuel

27 First" or "OFF" principle.

28 **ORDER RE SUMMARY JUDGMENT-**   **621**

factors of 1.32 and 1.64 respectively. (Klementiev 1997

Affidavit at p. 4).

Defendants contend that what Klementiev has done is simply

multiply HEDR's results. Although Klementiev asserts it is not

that "simple," he essentially concedes it is a multiplication

process:

> I have made a natural suggestion: If both MERI+
> and the HEDR model behave similarly when FIFO was
> not violated they should behave similarly when
> FIFO was violated.
>
> Therefore, one has to expect the HEDR model output
> would increase by 32% if the HEDR model was run under
> the suggestion that FIFO was violated.

(Klementiev 1997 Affidavit at pp. 4-5).

The results of Klementiev's "adjustments" are found in Table

7.1 of his 1995 report (pp. 100-101). Scenario 1 is HEDR's

monthly estimates. (Klementiev 1995 Report at p. 97). Scenario

2a assumes one bucket (of slugs) out of every eight buckets was

transposed between 1944 to 1949, except for certain periods

during which Klementiev says there were no FIFO protocol

violations. (Id. at p. 98). Comparing the Scenario 1 HEDR

figures to the Scenario 2a figures, one can see they are

identical for the months December 1944 through April 1946, August

1947 to December 1948, and December 1949. For all of the other

months, May 1946 to July 1947 and January 1949 to November 1949,

Klementiev increases HEDR's monthly estimates by 64%.

Klementiev's total I-131 release estimate under Scenario 2a is

752,048 curies, an approximate 8% increase over HEDR's estimate

of 695,971 curies. (Klementiev 1995 Report at p. 113, Table

ORDER RE SUMMARY JUDGMENT-   622

7.6).

Klementiev's Scenario 2b assumes half a bucket (of slugs)
out of every eight buckets was transposed during the **entire**
period of 1944-49. In Scenario 2b, Klementiev increases HEDR's
monthly estimates by 32%. (<u>Id</u>. at pp. 100-101, Table 7.1).   His
total I-131 release estimate under Scenario 2b is 918,812 curies.
(<u>Id</u>. at p. 113, Table 7.6).

The issue here is whether either of these "adjustment"
factors and the estimates they produce are scientifically
reliable (methodologically sound).

(3)   Reliability of Klementiev's Analysis

(a)   Data Used to Arrive at Adjustment Factors

Klementiev arrived at his "adjustment" factors by examining
the first seven reactor discharges or "pushes" out of a total of
226 reactor discharges which occurred between 1944 and 1947,
otherwise known as the Hanford "start-up" period. (Klementiev
Dep. at pp. 100-101; Klementiev 1995 Report at p. 82). Those
seven "pushes" involved 10 slug dissolutions (also referred to as
"dissolver charges") out of a total of 600 dissolutions or
charges which occurred between 1944 and 1947. (Klementiev Dep.
at p. 101). Those 10 dissolutions were the second (No. 10002)
through eleventh dissolutions (No. 20011) processed through
Hanford. (Klementiev Report at p. 80, Table 5.4). According to
defendants, dissolver charge no. 10002 involved slugs discharged
from the reactors on November 24, 1944 and dissolver charge no.
20011 involved slugs discharged from the reactors on March 22,

ORDER RE SUMMARY JUDGMENT-   623

1   1945. Neither plaintiffs' counsel or Klementiev dispute those

2   dates.

3   According to Klementiev:

4   1960 Ci per charge was dissolved in average
             during the period when the charges from 10002
5   to 20011 were processed. If just one bucket
             per charge were allowed to be transposed then
6   in average additional 1,250 Ci per charge would
             be dissolved. It means that if one bucket per
7   charge was transposed then the average estimate
             of the additional I-131 activity is about 64%
8   of the total dissolved I-131 activity.

9   (Klementiev Report at p. 82).[499] Put another way, rather than

10 HEDR's average figure of 1960 Ci per charge for this period, a

11 one bucket transposition would raise that average figure to 3,210

12 Ci per charge. This is an increase of 64% (1,960 + 1,250 =

13 3,210).[500]

14   Defendants contend Klementiev's selection of this limited

15 data is unscientific. They say Klementiev has no idea about the

16 impact of his assumption (one bucket transposed per charge) for

17 _____

        [499] Each bucket holds 105 slugs. A nominal dissolver charge
18 or "batch" is eight buckets. Eight buckets is the equivalent of
    840 slugs or 3.3 tons (7.85 lb per slug). (Heeb 1993 Vol. 1 at
19 p. 1.6).

20   [500] Plaintiffs' expert, Dr. Kenneth McNeill, provides this
    example in his November 1995 report:
21
            [C]onsider a series of batches of slugs which
22   have all had different cooling times to this
            minute, and have activities in the ratios 1:2:
23   4:8. If they are dissolved at 8 day intervals
            in the above order, the [iodine] release will be
24   1 + 1 + 1 + 1 = 4. If however order is, by mistake,
            changed, so that the fourth one is dissolved first,
25   the release will be 8 + 0.5 + 0.5 + 0.5 = 9.5.
            Even if simply the second and third are interchanged,
26   the release is 1 + 2 + 0.5 + 1 = 4.5.

27 (Foulds' Ex. 74 at p. 4).
28
    ORDER RE SUMMARY JUDGMENT-   624

1   periods **later than March 1945**, when dissolutions occurred with

2   more frequency. According to defendants, because dissolutions

3   occurred with more frequency after March 1945, "the differences

4   in the cooling times between dissolutions, and hence the amounts

5   of iodine-131 released given a transposition from one dissolution

6   to another would be lessened." In other words, because the

7   frequency of dissolutions was less for the period before March

8   1945, a transposition (newer fuel being dissolved before older

9   fuel) would result in a higher average amount of I-131 released.

10 This is because of the greater **disparity** in the cooling times

11 between the transposed "older" fuel and the "newer" fuel and

12 therefore, the greater disparity in the iodine content between

13 each.[501]

14   As an example of the relative infrequency of dissolutions

15 prior to March 1945, defendants note that seven of Klementiev's

16 dissolutions occurred at T-Plant, six of which occurred during a

17 72 day period between December 26, 1994 and March 8, 1945. This

18 is an average of only one dissolution every 12 days. (Klementiev

19 1995 Report at p. 80, Table 5.4).[502]

20 _____

21   [501] The "newer" fuel has greater iodine content because it
    has not cooled as long. The "older" fuel has less iodine content
22 because it has cooled a longer period of time. The longer the
    "older" fuel sits before dissolution, the more it has cooled and
23 the less iodine content it has. When dissolutions are less
    frequent, the "older" fuel sits even longer in the transposition
24 scenario, widening the disparity between its iodine content and
    that of the "newer" fuel which gets dissolved first in the
25 scenario.

26   [502] Dissolver charge no. 10001 occurred on dissolving date
    92. Dissolver charge no. 10007 occurred on dissolving date 164.
27 (164-92 = 72).

28 **ORDER RE SUMMARY JUDGMENT-   625**

When the frequency of dissolutions is increased, the **disparity** between the cooling times of the transposed "older" fuel and "newer" fuel is lessened as is the respective iodine content of each.[503] Therefore, a one bucket transposition does not produce as a great a percentage increase in the average amount of iodine released. Defendants note that in October 1945, 17 dissolutions occurred in a 31 day period at the T-Plant, averaging out to one dissolution every **second day**. (Heeb 1993 Vol. 2 at pp. 6.7-6.9, Table 6.1). Thus, instead of the 64% increase Klementiev arrived at based on his analysis of reactor discharges from November 1944 to March 1945, defendants suggest the percentage increase could well have been less for the period **after** March 1945.

Klementiev acknowledges that possibility as well. Asked whether his 64% increase applied to all periods, he said that was "not necessarily" so and the final estimate of the release could be "higher or lower" for other periods depending on the sample taken. (Klementiev Dep. at p. 98-99). He also conceded his analysis of the second through eleventh dissolver choices was "probably not the best choice." (<u>Id</u>. at p. 93).

In his affidavit, Klementiev does not appear to change his position. He says the defendants' argument is a concession that FIFO violations must be accounted for and contradicts their position that such violations did not occur. According to

_____

[503] When the dissolutions are more frequent, the "older" fuel does not sit as long before dissolution. Therefore, there is not as great a disparity in iodine content as between it and the "newer" fuel which gets dissolved first.

ORDER RE SUMMARY JUDGMENT-   626

Klementiev, he agrees that considering the first 100 dissolutions "would be a **better choice** than the first 10 chosen in my report." He adds that if the first 100 were used for averaging, "it does not necessarily mean that the resulting average would be lower." Klementiev says the defendants themselves could have calculated an alternative average based on additional dissolutions, had they chosen to do so, and that this would have been "technically very easy." (Klementiev 1997 Affidavit at p. 7).

Defendants do not assert FIFO violations never occurred or could never have occurred. Rather, they take issue with the basis for Klementiev's conclusion that they occurred as often as assumed by his Scenario 2a and Scenario 2b. Secondly, this motion in limine is directed at Klementiev and therefore, he has to justify the methodological soundness of his decision not to consider additional dissolutions for which historical data (logbooks) was available. Klementiev cannot pass that burden on to defendants' counsel. Because there are logbooks covering the period December 1944 to April 1946[504], August 1947 to December 1948 and December 1949, Klementiev cannot say he lacked the data to analyze other dissolutions. Those logbooks showed no FIFO violations for those months and hence, that is the reason why Klementiev's Scenario 2a estimates for those months are **identical** to HEDR's estimates (Klementiev's Scenario 1). (Klementiev Dep. at p. 63).

---

[504] This period includes what defendants assert is the critical period after March 1945 when dissolutions were more frequent.

ORDER RE SUMMARY JUDGMENT-   627

1   In their brief, plaintiffs' counsel advance essentially the

2 same argument as Klementiev does in his affidavit. Like

3 Klementiev, counsel argue that "defendants merely suggest,

4 without offering any proof, that the correction factor Klementiev

5 derived from [his] transposition exercise actually 'inflates' his

6 iodine estimates and that modeling the first 10 dissolvings

7 'ensured even more potential error. . . .'" They contend that

8 because defendants have Klementiev's equations, "they should have

9 tested it empirically" based on other dissolutions for other time

10 periods and "included the results in their motion." Plaintiffs

11 suggest defendants did not do this because they feared the result

12 would be more than a 64% increase.

13   Plaintiffs' counsel assert that if Klementiev had chosen

14 October 1945 because of the more frequent dissolutions during

15 that month, it "would have yielded Klementiev an even more

16 'inflated' correction factor had that been his intent . . . ."

17 Plaintiffs say defendants overlook the fact that a campaign was

18 started in September 1945 for the discharge and sorting of Class

19 "C" fuel, a higher "burnup" fuel containing much higher levels of

20 iodine at discharge from the reactor and therefore, assigned

21 longer cooling periods. According to plaintiffs, an inadvertent

22 transposition between Class "C" fuel and Class "A" fuel would

23 have resulted in a greater "correction factor" or average because

24 of the disparity in cooling times between the two types of fuel.

25 Plaintiffs say Klementiev modeled the "start up" period because

26 the fuel (uranium slugs) had comparable "burnups" and comparable

27 cooling times "which would bias the correction factor low in

28 **ORDER RE SUMMARY JUDGMENT-   628**

contrast to a transposition exercise with a mixture of high- and low-burnup fuel."

This is **not** an argument advanced by Klementiev himself as justification for his use of the "start up" period dissolutions. The fundamental question is whether as part of the scientific method, **Klementiev** should have considered other dissolutions in assessing the accuracy of HEDR's iodine estimates. **Klementiev** has to justify why he did not consider those other dissolutions, for which historical records were in fact available. It **may** be that for any number of reasons the "correction factor" or average would be higher than 64% for periods other than the "start up" period. That is beside the point. This court is concerned with **methods**, not **conclusions**. Klementiev does not provide a **conclusion** based on other dissolutions because his **method** did not include analysis of those other dissolutions.

Although Klementiev maintains his use of just the ten dissolutions during the "start-up" period is scientific, he also stated at his deposition that "if I were your boss I wouldn't allow you to manipulate with ten only [and] I would say I will give you more time, more money and I would say go with a hundred." (Klementiev Dep. 106-07). Time and money can never be an excuse for insuring the reliability of results. This is a very revealing comment from Klementiev about the soundness of his methodology.

(b)   FIFO Assumptions

Defendants contend Klementiev makes erroneous, critical

ORDER RE SUMMARY JUDGMENT-   629

1 assumptions about violations of the FIFO principle, namely: 1)

2 that HEDR assumed compliance with FIFO in analyzing iodine

3 releases for the 1944-49 period; and 2) Hanford's operators

4 "regularly" violated the FIFO principle during the 1944-49

5 period.

6   Section 2 of Klementiev's 1995 report (pp. 23-26) is devoted

7 to what he believes is evidence from which "it is reasonable to

8 **suggest** that FIFO protocol was violated **regularly** throughout the

9 Hanford Works history." (Emphasis added). Klementiev ultimately

10 concludes that "documented evidence shows FIFO was violated **or**

11 **likely could be violated** in the real dissolving practice."

12 (Klementiev 1995 Report at p. 76)(Emphasis added).

13

14   **(i)   Direct Evidence of FIFO Violation**

15   Klementiev states there is "direct evidence" of a FIFO

16 violation having occurred in June 1946, citing Jaech J.L,

17 "Monthly Summary of Dissolver Data- 12/31/44 through 6/30/57,"

18 FTS-CLVI-73, General Electric Company, Hanford Works, Richland,

19 Washington.[505] According to Klementiev, fuel from "pushes"

20 (reactor discharges) on January 18 and January 20, 1946 was

21 processed in June 1946 **after** processing of fuel from the "pushes"

22 of February 26, March 12 and the first portion of the "push" of

23 March 17, 1946. (Klementiev 1995 Report at pp. 23-24).

24   Defendants describe the June 1946 processing as involving

25 the dissolution of "a small quantity of uranium fuel that had

26 _____

27   [505] Hereinafter, "Jaech." (Defendants' Ex. 57).

28 **ORDER RE SUMMARY JUDGMENT-   630**

cooled for **163 days** when the prevailing "cooling time" was **60 days**. At his deposition, Klementiev indicated the "cooling time" for the January 18 and January 20 "pushes" was 131 and 133 days respectively and that the normal cooling time was between 60 and 90 days. (Klementiev Dep. at p. 140).[506] There is no dispute that a "transposition" occurred in June 1946 when "newer" fuel, cooled for the standard period of time (60 or 90 days), was dissolved before "older" fuel which had cooled for a longer period of time.

Defendants point out that HEDR considered the specific 1946 situation in its "batch-by-batch" analysis and modeled the incident as it actually occurred, rather than according to the FIFO assumption. (Heeb 1993 Vol. 2, Table 6.1 at p. 6.18). Klementiev begins Section 2 of his 1995 report by stating that "Oldest-Fuel-First (or, First-In-First-Out: FIFO) suggestion was **used** in [HEDR] for reconstruction of the fuel processing schedule" and that "[t]his suggestion plays important role in the reconstruction of radioiodine releases occur[ring] in the period from 1944 to 1949." (Klementiev 1995 Report at p. 23) (Emphasis added). Defendants contend this statement shows Klementiev errantly assumed HEDR **always** based its iodine emission estimates solely on the amount of iodine present in the oldest slugs available for dissolution.

When informed at his deposition that HEDR had considered the

---

[506] See discussion _infra_ which indicates Klementiev was short 30 days regarding the cooling time for the January 18 and 20, 1946 "pushes."

ORDER RE SUMMARY JUDGMENT—   631

1 fuel sequencing variation of June 1946, Klementiev asserted that

2 "in general" HEDR followed the "FIFO suggestion." He maintained

3 that he had "never said that HEDR didn't model [the June 1946]

4 situation."   (Klementiev Dep. at p. 141-143). According to

5 Klementiev:

> The only thing I stress here, that violation
> of FIFO took place. Now, if you tell me
> that in this particular case HEDR considered
> this particular situation, it doesn't mean
> that HEDR did it always, and I can show you
> cases or time periods or pushes - - and I do
> remember I found them - - when HEDR ignored
> that.

(Id. at p. 143).

Klementiev added that the "next" step in the analysis would

be the impact of HEDR's consideration of particular FIFO

violations. Although Klementiev asserted he had taken this

"next" step, he acknowledged it was "probably not" in his report.

(Id. at p. 151).

The FIFO assumption is the basis for Klementiev's challenge

of HEDR's 1944-49 iodine release estimates. Failure to follow

FIFO is the reason for Klementiev's assertion that one out of

every eight buckets of uranium slugs was transposed, resulting in

newer fuel being dissolved before older fuel. Consequently, if

HEDR accounted for FIFO violations ("modeled" them), that

undermines Klementiev's basis for challenging HEDR iodine release

estimates. Therefore, an important question is whether

Klementiev actually identified other FIFO violations not

accounted for by HEDR.

With regard to the June 1946 transposition, defendants note

ORDER RE SUMMARY JUDGMENT-   632

that although older fuel was dissolved before newer fuel, both the older and newer fuel were subject to the "standard" cooling time of 60 days. It appears the older fuel from the January 18 and 20, 1946 "pushes" was cooled for 163 and 161 days respectively prior to dissolution in June 1946.[507] It appears the "newer" fuel from the more recent "pushes" (February 26, March 12 and March 17, 1946) was cooled for 94, 80, and 75 days respectively prior to dissolution in May 1946.[508]

At his deposition, Klementiev testified the "impact" of this transposition was actually "positive." The iodine release is lower when a transposition of older and newer fuel occurs where **both** have been cooled for at least the standard cooling time, with the "older" fuel obviously having cooled for an even longer period of time. (Klementiev Dep. at p. 46). Therefore, although technically an FIFO violation occurred in June 1946, it was of no consequence.

//

---

[507] Table 2.1 of Klementiev's 1995 Report at p. 24 is "Monthly Summary of Dissolver Data" compiled from the Jaech document. The January 18 "push" occurred on day number 480 from the D Reactor. It was dissolved on day number 643 (643-480=163). The January 20 "push" occurred on day number 482 from the F Reactor. It also was dissolved on day number 643 (643-482=161).

[508] The February 26 "push" occurred on day number 519 from the D Reactor. It was dissolved on day number 613 (613-519=94). The March 12 "push" occurred on day number 533 from the D Reactor. It was dissolved on day number 613 (613-533=80). Part of the March 17 "push" occurred on day number 538 from the B Reactor. It too was dissolved on day number 613 (613-538=75). All of these dissolutions occurred in May 1946, thirty days prior to the dissolutions of the January 18 and January 20 "pushes" in June 1946. The dissolutions of the January 18 and 20 "pushes" occurred on day 643 (643-613=30). (Table 2.1 of Klementiev's 1995 Report at p. 24).

ORDER RE SUMMARY JUDGMENT-   633

(ii) Circumstantial Evidence

Plaintiffs contend HEDR used the FIFO assumption **where**

dissolving records are incomplete and with the exception of the

1963 PUREX release, ignored inadvertent variations in fuel

sequencing for all time periods. They note departures from FIFO

were not among the variables considered by HEDR in determining

uncertainty in dissolver operations and iodine content in "STRM."

(Heeb 1993 Vol. 1 at p. 4.13). Plaintiffs assert that "one

documentary basis" for considering this "parameter" (violations

of FIFO) is Jaech (cited <u>supra</u>).

In addition to using Jaech to point out the specific June

1946 FIFO violation, Klementiev's 1995 report had this to say:

> Of course, FIFO violation **could** have happened
> not only within time frame of two consecutive
> months of processing. We may expect that FIFO
> violation could have happened within any given
> month, though it is not evident from the month's
> records in [Jaech] where the records are sorted
> out by ascending date of push.

(Klementiev 1995 Report at p. 17)(Emphasis added). According to

plaintiffs, what this means is that Jaech is not a "complete"

document because it only identifies the month, rather than the

day of dissolving. Hence, one cannot look at Jaech and say with

certainty that other FIFO violations occurred. Nevertheless,

Klementiev asserts that the lack of completeness means departures

from FIFO "could" have occurred within any given month.

Obviously, Klementiev uses Jaech to hypothesize that

additional FIFO violations occurred. Jaech itself is not proof

that additional violations **in fact** occurred. The plaintiffs seem

to recognize as much, and therefore argue that Klementiev's

ORDER RE SUMMARY JUDGMENT-   634

hypothesis is borne out by what they say are historically

documented FIFO violations, deliberate and inadvertent.

Klementiev discusses these in his report at Section 2.1,

"Inadvertent Shipment of Green Fuel," and Section 2.3, "200 North

Area Shipments." (Klementiev 1995 Report at pp. 23-26).

Plaintiffs contend records of the "200 North Area

Shipments," (HAN-45801)[509], show deliberate departures from FIFO

protocol. In his report, Klementiev presents in a table (Table

2.2) the schedule of fuel (slug) shipments from the 212 P and 212

R Buildings. According to Klementiev, "[t]his fuel was **normally**

dissolved soon after shipment, and therefore the schedule of the

shipments is **about the same** as the schedule of [dissolver] runs

with **some reasonable time gap**." (Klementiev 1995 Report at p.

24) (Emphasis added). Klementiev states that Table 2.2 shows

clear FIFO violations:

> For example, one can see from the 212 P
> Building records that fuel from the push
> "12-3-1946" was shipped for dissolving after
> shipping the "greener" fuel from the push
> "12-11-1946", and also fuel from the push
> "12-17-1946" was shipped after shipping fuel
> from the push "12-26-1946."
>
> If the shipments from 212 P Building were compared
> with shipments from 212 R Building, one could see
> that the fuel discharged from D Reactor at "12-11-
> 46" was shipped to B-Plant at "2-17-47." However,
> the other portion of the fuel that was discharged
> from the D Reactor **later**, at "12-17-46," was shipped
> to same B-Plant four days **earlier**, at "2-13-47."

(Id. at p. 25)(Emphasis in text).

_____

[509] Also known as "Compilation of 200 Area Monthly
Production Reports, January-June 1947," (July 1947). Foulds' Ex.
38.

ORDER RE SUMMARY JUDGMENT-   635

1   Defendants emphasize that these are merely "shipping"

2 records which show when irradiated uranium fuel slugs were

3 shipped to the separations plants. Defendants argue the fact

4 that slugs were **shipped** out of order (newer fuel **shipped** before

5 older fuel) does not necessarily mean they were **dissolved** out of

6 order.

7   Plaintiffs contend defendants' own documents, specifically

8 the "Hanford Engineer Works Technical Manual," (May 1, 1944) (HW-

9 10475)[510], shows that dissolving "routinely" commenced "shortly"

10 after shipping from the 212 Area. They point out that Section C

11 of this manual at p. 312 states metal (slugs) coming into the

12 Canyon Building (separations plants) from the 212 Storage Area

13 "is charged directly from the cask car to the dissolvers, except

14 in the case of buckets containing damaged slugs coming in for

15 storage." Plaintiffs assert that because of the hazards

16 involved, as described in the technical manual, fuel was simply

17 not left lying around in cask cars.

18   One can argue this is persuasive circumstantial evidence

19 that shipping dates coincided closely with dissolving dates, and

20 hence that the **specific** "out-of-order" shipping dates identified

21 by Klementiev from the 200 North Area shipping records suggest

22 FIFO violations could have occurred with regard to those

23 **particular shipments**. The defendants do not dispute the

24 plaintiffs' arguments based on the technical manual.

25   Plaintiffs also cite the "200 Area Daily Log Book," (HAN-

26

_____

27   [510] Foulds' Ex. 39.

28 **ORDER RE SUMMARY JUDGMENT-   636**

1 45761)[511], which they say constitutes further evidence that

2 dissolving routinely commenced within a day of shipping.

3 According to plaintiffs, this log book shows that two FIFO

4 violations occurred in September 1945, specifically that fuel

5 from the August 16, 1945 "push" was shipped for dissolving before

6 the remaining fuel from the August 9 "push," and fuel from the

7 August 17 "push" was shipped before the remainder of the fuel

8 from the August 9 and August 16 "pushes."   (Plaintiffs' Br. at

9 pp. 20-21).

10   One problem, however, is that plaintiffs do not say where in

11 **Klementiev's** report one can find that he used the technical

12 manual or the 200 Area daily log book to assert that dissolving

13 routinely occurred "shortly" or "within a day" of shipping.

14 Klementiev was much more equivocal about the timing of shipping

15 and dissolving: "This fuel was **normally** dissolved **soon** after

16 shipment, and therefore the schedule of the shipments is **about**

17 **the same** as the schedule of runs with some reasonable time gap."

18 Furthermore, Klementiev does not identify FIFO violations in

19 September 1945. It is plaintiffs' **counsel**, and **not** Klementiev,

20 who have dug up this particular example from the "200 Area Daily

21 Log Book." Indeed, Klementiev's Scenario 2a assumes there were

22 **no** FIFO violations for the time period between **December 1944 and**

23 **April 1946.** Klementiev agrees with HEDR that the logbooks show

24 no FIFO violations for that period of time.

25   More importantly, as will be discussed later, one cannot

26

27   [511] Foulds' Ex. 114.

28 **ORDER RE SUMMARY JUDGMENT-   637**

1 forget the large assumptions underlying Klementiev's analysis.

2 If Klementiev has in fact identified FIFO violations for the

3 specific dates identified by him in December 1946 and February

4 1947 based on the 200 North Area shipping records, is that enough

5 of a foundation to support his Scenario 2a assumption that one

6 out of every eight buckets was transposed, or his Scenario 2b

7 assumption that one-half bucket out of every eight buckets was

8 transposed?

9   As an example of what he labels an "inadvertent shipment of

10 green fuel," Klementiev quotes from Keene, A.R., "Separations

11 Section Radiation Monitoring Monthly Report, September 1954,"

12 (October 1, 1954)(HW-33246)[512]:

13   [A]lthough the possibility of an inadvertent
     shipment of green metal feed could not be
14   definitely determined, there was sufficient
     evidence throughout the process to accept this
15   explanation for the uncontrolled iodine emission
     which occurred through the latter part of
16   August [1954] and the first part of this month
     [September 1954].

17
(Klementiev 1995 Report at p. 23).

18
  According to Klementiev, the metal could only be

19 unexpectedly green if the cooling time was unexpectedly short and

20 this was unknown prior to dissolving. Under these circumstances,

21 says Klementiev, FIFO could not be properly implemented.

22 Furthermore, Klementiev maintains it was reasonable to suggest

23 that this problem existed as much or more during the first years

24 of the Hanford Works when technical staff had less technological

25 experience than in 1954. (Id.). While defendants do not dispute

26 —————————————————————

27   [512] Foulds' Ex. 69.

28 **ORDER RE SUMMARY JUDGMENT— 638**

1 that FIFO violations may have occurred in August and September

2 1954, they point out that the period under examination is **1944-**

3 **49.**

4   The plaintiffs argue that "even though [the defendants] and

5 the government destroyed many of the historical documents from

6 the early Hanford era, defense counsel would require that

7 Klementiev document every FIFO violation down to the very bucket

8 or slug that was 'mixed up.'" According to plaintiffs, the

9 absence of such detailed proof did not prevent defendants,

10 specifically General Electric, from concluding that green fuel

11 was the cause of releases in August and September 1954.

12 Plaintiffs' counsel say a review of T-Plant Metal Histories for

13 late August 1954 confirms that newer fuel was dissolved before

14 older fuel. "T-Plant Percent Book (1953-1955)" (FTS-XX-

15 1658).[513] However, any such review was undertaken **not** by

16 Klementiev, but by plaintiffs' counsel.

17   In their brief, plaintiffs cite a 1964 document, "Activity

18 of Irradiated Regular Metal In Buckets" by R.H. Smith, (HW-

19 84001).[514] According to plaintiffs, this documents shows

20 General Electric concluded inadvertent shipping of green fuel was

21 a "common problem to all Reactor Processing Operations."

22 Actually, the document says "[t]he **potential** of shipping a bucket

23 of metal that has not had sufficient decay time is a **common**

24 problem to all Reactor Processing Operations." (Smith 1964 at p.

25 _____

26   [513] Foulds' Ex. 31.

27   [514] Foulds' Ex. 103, hereinafter "Smith 1964."

28

ORDER RE SUMMARY JUDGMENT-   639

2). That there is a "potential" problem does not mean there is

an "actual" problem which is a "common" occurrence. Furthermore,

Smith is a 1964 document and the focus here is the 1944-49 time

period.

D.E. Cooley, "Irradiated Fuel Age Determination Study" (HW-

83869)[515], is also a 1964 document (August 31, 1964). In a

footnote at p. 98 of his 1995 report, Klementiev quotes from this

document as providing justification for his Scenario 2a which

assumes one out of every eight buckets was transposed. The quote

from Cooley 1964 at p. 2 is as follows:

> **From time to time** a quantity of incompletely
> aged irradiated fuel has been sent by mistake
> from the reactors to the processing areas. The
> release of uncontrollable quantities of harmful
> fission products such as I-131 upon processing
> this fuel creates a need for a method to prevent
> its shipment to the processing areas. At present
> this is done by procedural techniques using
> cards, filing techniques, and cross checks. These
> methods are entirely independent of the characteristics
> of the fuel itself and are subject to failure
> from human error. The **occasional** shipments of fresh
> fuel attest to the weakness of the procedural
> method. It is therefore advantageous to find a method
> of determining the age of irradiated fuel after
> discharge from the properties of the fuel.

(Emphasis added).

At his deposition, Klementiev acknowledged that "from time

to time" and "occasional" do not mean the same thing as

"regularly." (Klementiev Dep. at p. 40). Cooley 1964 also again

raises the issue of whether a **shipment** of green fuel to the

processing area means the shipment was in fact **dissolved** ahead of

older fuel.

---

[515] Defendants' Ex. 19.

ORDER RE SUMMARY JUDGMENT-   640

1   **(c) Errors Regarding December 1944 and January 1949**

2   **Estimates**

3   According to defendants, because Klementiev's 1944-49

4   estimate was not derived by the "detailed batch-by-batch"

5   approach used by HEDR, the "poverty" of his approach is

6   manifested by his admission that his December 1944 and January

7   1949 monthly estimates are miscalculated. The focus here is on

8   Klementiev's 2b Scenario because, as noted previously,

9   Klementiev's Scenario 2a estimates for December 1944 and January

10 1949 are identical to HEDR's estimates for those months. As

11 acknowledged by Klementiev, the logbooks did not show any FIFO

12 violations for December 1944 to April 1946 and for December 1949.

13 For his Scenario 2b, Klementiev increases HEDR's estimates by 32%

14 across the board for each and every month.

15   At his deposition, Klementiev was asked if his Scenario 2b

16 estimate for December 1944 (2,823 Ci) could be correct if it was

17 based on an assumption that one bucket of fuel (slugs) from the

18 first dissolving batch was transposed with an earlier dissolving

19 batch. There were no dissolving batches prior to December 1944.

20 Klementiev stated that his December 1944 estimate could not be

21 correct if the **"transposition** is suggested" and added that

22 **"transposition** may not be considered here." (Klementiev Dep. at

23 p. 71)(Emphasis added). He tried to provide an alternative

24 explanation, but was unsuccessful:

25   Now, as for this particular month, I would explain
            this difference in cooling time **not in terms of**
26   **transposition** but in terms of it could be that some
            of the fuel was residing longer in the reactor, which
27   probably doesn't explain much because it was
28

ORDER RE SUMMARY JUDGMENT-   641

1 saturated situation anyway. How I would explain
   for myself is definitely residing in the reactor
2 wouldn't explain this. As for now, I couldn't give
   you proper explanation for that and I have to think
3 again about why I included this particular month
   in the calculation. That's very special month and
4 I agree with that.

5 (Id. at p. 72)(emphasis added).

6   In their brief, plaintiffs' counsel try to fill the gap left

7 by Klementiev. Although Klementiev testified that transposition

8 of buckets could **not** be considered as a basis for increasing his

9 December 1944 estimate, plaintiffs' counsel contend that a

10 transposition was, in fact, "possible." This is counsels'

11 explanation:

12   The buckets of fuel were loaded from the
            reactor discharge basin into the cask car,
13   transported to the 212 North Area where the
            buckets were unloaded into the 'intermediate'
14   storage areas, and then reloaded onto the
            cask cars when the fuel was ready for shipping
15   to be dissolved at B or T Plants. The trans-
            positions **must** occur **before** the metal reaches
16   the separations plants! Therefore, a transposition
            was possible and properly considered by Klementiev
17   where 14.2 tons of fuel from 3 distinct reactor
            pushes (11/4, 11/24 and 12/20/44) were available
18   for the initial 3.3 ton dissolving on 12/26 . . . .

19 (Plaintiffs' Response Br. at p. 25, citing Heeb 1993, Vol. 2 at

20 Table 6.1, p. 6.2)(Emphasis in text).[516]

21   In other words, counsel suggest a bucket of slugs from the

22 12/20/44 "push" **could** have been transposed with a bucket from the

23 11/6/44 "push," or fuel from the 12/20/44 "push" **could** have been

24 shipped to the separations plant before fuel from the 11/6/44

25 _____

26   [516] It appears that actually 3.5 tons was dissolved on
   12/26/44 and that the earliest "push" occurred on 11/6/44 rather
27 than 11/4/44.
28
   ORDER RE SUMMARY JUDGMENT-   642

"push." This is pure speculation by **counsel** and it is not part

of **Klementiev's** rationale. Table 6.1 of Heeb 1993 Vol. 2 shows

the 12/26/44 dissolution involved three dissolver cuts from two

"pushes," the 11/6/44 "push" and the 11/24/44 "push." The slugs

from both these "pushes" had been cooled for at least thirty days

which apparently was the standard cooling period at that time.

There is nothing to indicate buckets from the 11/6 and 11/24

"pushes" were transposed.

In December 1949, the "Green Run" was conducted involving a

deliberate release of approximately 7,000 Ci of I-131 into the

atmosphere. Klementiev testified that according to his

knowledge, these releases occurred from the dissolution of slugs

which had been cooled for only 16 days. Klementiev was asked

what the difference would be between the effect of his assumed

transposition of buckets versus the effect of a 16 day cooling

period. Klementiev responded that he had made an error, that he

would not allow his model to calculate the "transposition" effect

for December 1949, that he had failed to consider this "special

situation," and that he would discard his Scenario 2b estimate

which increased HEDR's estimate by approximately 2,300 curies.

(Klementiev Dep. at pp. 73-74). In other words, Klementiev

conceded the accuracy of HEDR's December 1949 estimate (7,241 Ci)

which took into the account the 16 day cooling time, and conceded

there was no need to increase that amount to 9,558 Ci as he had

done. (See Table 7.1 at p. 101 of Klementiev 1995 Report).

Defendants assert these mistakes could have been avoided had

Klementiev conducted a detailed batch-by-batch review of

ORDER RE SUMMARY JUDGMENT-   643

1 historical operations. They contend it would have been difficult

2 for Klementiev to do such a review because of his admission that

3 he did not how the buckets were "queued" or lined up mechanically

4 within the cooling basins, (Klementiev Dep. at pp. 136-37), and

5 had never seen any of the reactors, storage buildings or

6 separations plants. (Id. at p. 135; 215-16).

7

8   (d) Summary

9   Klementiev concedes the accuracy of the historical records

10 regarding dissolving operations. It is for that reason, his

11 Scenario 2a does not propose to increase HEDR's monthly estimates

12 for the following time periods: December 1944 to April 1946;

13 August 1947 to December 1948; and December 1949. Historical

14 records exist for those time periods and no FIFO violations are

15 shown. Historical records do not exist for the period between

16 May 1946 and July 1947, and between January 1949 and December

17 1949. For those periods, Klementiev's Scenario 2a increases

18 HEDR's release estimate by 64%. In sum, Klementiev seemingly

19 asserts his analysis did not require him to conduct a detailed

20 batch-by-batch review of historical operations. The critical

21 question therefore is whether he has a reliable scientific basis

22 for assuming that one out of every eight buckets of slugs was

23 transposed during those periods for which no historical records

24 exist.

25   Scenario 2b assumes a half-bucket transposition occurred

26 during the handling of **each and every** batch of slugs during **each**

27 **and every month** between 1944 and 1949. This is despite

28 **ORDER RE SUMMARY JUDGMENT-   644**

Klementiev's concession that historical records show no FIFO

violation for December 1944 to April 1946; August 1947 to

December 1948; and December 1949. According to Klementiev,

because of the "daily data" he was not allowed to assume an FIFO

violation for those periods. (Klementiev Dep. at pp. 64-65).

Indeed, Klementiev testified that as between Scenarios 2a and 2b,

Scenario 2a was "more probable." (Id. at pp. 373-74). Based on

this statement from Klementiev, defendants contend Scenario 2b is

not an issue.

Plaintiffs disagree and it is understandable why they do.

Plaintiffs' other experts, Dr. Stewart (dispersion of iodine in

the environment) and Dr. Crawford-Brown (dose estimation) rely on

the Scenario 2b estimate. However, the best explanation

plaintiffs can offer is that "both scenarios are reliable in the

sense that [Klementiev's] model creates results that reliably

reflect the input data" and "which scenario utilizes the **least

number of assumptions**, which . . . was 2a." According to

counsel, if one accepts the premise that available batch

dissolving records are correct, then Scenario 2a becomes more

certain. On the other hand, if one does not accept that premise,

Scenario 2b becomes more certain.

What plaintiffs essentially contend is that the documents

cited by Klementiev (and those cited by counsel) as evidence of

FIFO violations are sufficient to call into question the accuracy

of the historical dissolving batch records, and therefore it is

scientifically reasonable to assume a half-bucket transposition

per batch for each and every month in the 1944-49 time period.

ORDER RE SUMMARY JUDGMENT-   645

1 (Klementiev 1997 Affidavit at pp. 17-18).

2   Obviously, the problem is that Klementiev testified there

3 was no reason to quibble with the accuracy of the dissolving

4 records:

5   HEDR suggested no violations and I agree with
           that because we have, both HEDR and I, we have
6   exact records pertaining to each dissolving.

7 (Klementiev Dep. at p. 63).

8   Klementiev's statement, by itself, is a sufficient basis for

9 finding the higher Scenario 2b estimate to be wholly speculative

10 and therefore, scientifically unreliable.

11   This leaves the lower Scenario 2a estimate and the critical

12 question of whether Klementiev has a reliable scientific basis

13 for assuming that one out of every eight buckets of slugs was

14 transposed during the periods for which historical dissolving

15 records **do not** exist. That depends on the documents cited by

16 Klementiev in his report and which have been discussed above.

17   **Klementiev** himself provides direct evidence of but one

18 documented instance of a FIFO violation in June 1946. As it

19 turns out, that "violation" is inconsequential and indeed,

20 lessens the overall amount of I-131 released. This is because

21 the "newer" fuel had been cooled for at least the standard period

22 of time (60 to 90 days), while the "older" fuel had been cooled

23 significantly in excess of the standard period of time (163 and

24 161 days).

25   Other than the June 1946 violation, Klementiev relies on

26 circumstantial evidence suggesting other FIFO violations

27 occurred. This evidence suggests: 1) the possibility of an

28

ORDER RE SUMMARY JUDGMENT-   646

1 inadvertent shipment of green metal feed in September **1954**; 2),

2 "occasional" shipments at some unspecified time of "incompletely

3 aged irradiated fuel" in an unspecified quantity and of an

4 unspecified age; and 3) several shipments of fuel from the 200

5 North Area occurred out of order between December 1946 and

6 February 1947, suggesting a likelihood those shipments were

7 dissolved out of order. As noted, the latter is arguably

8 persuasive circumstantial evidence that FIFO violations occurred

9 with regard to those **particular shipments** in December 1946 and

10 February 1947.

11   Nonetheless, the court finds this circumstantial evidence

12 does not constitute a reasonable foundation for Klementiev's

13 hypothesis that **one out of every eight buckets** was transposed

14 between May 1946 and July 1947, and January 1949 and November

15 1949. Defendants note there were 600 dissolver batches between

16 1944 and 1947 (Klementiev Dep. at pp. 100-01), but that

17 Klementiev produces only one documented FIFO violation, that

18 occurring in June 1946.

19   Dr. Kenneth McNeill is a Professor of Physics at the

20 University of Toronto. He is the author of a November 10, 1995

21 "Report to Tom H. Foulds, Reference-Hanford Releases."[517] In

22 his report, McNeill states he "worked in a general consultative

23 and review manner with Dr. A.A. Klementiev in the production of

24 his reports on an Alternative Reactor Model and on Estimation of

25 Iodine Releases." McNeill offers only a very **general** and

26

_____

27   [517] Foulds' Ex. 74.

28 **ORDER RE SUMMARY JUDGMENT-**   **647**

1 **qualified** approval of Klementiev's overall work: "It is my

2 belief that the estimates given by . . . Klementiev . . . are,

3 **for the conditions and assumptions cited**, fair estimates of

4 [Iodine] emission."   (<u>Id</u>. at p. 8).

5   Klementiev's **assumption** that one out of every eight buckets

6 of slugs was transposed is not supported by an adequate

7 scientific foundation. McNeill recognized this. He informed

8 Klementiev in an April 28, 1995 E-Mail that "[e]verything would

9 be much tighter if you gave evidence that FIFO WAS violated."

10 (Defendants' Ex. 189 at p. 1)(Emphasis in text). McNeill raised

11 this concern at a couple of other points in his E-Mail: "Again,

12 one would love to see the evidence that FIFO was violated [and]

13 **[w]ithout it, one naturally does wonder whether the concern is**

14 **justified."** (<u>Id</u>. at p. 2)(Emphasis added).

15   The subject of an expert's testimony must be scientific

16 **knowledge.** "Scientific" implies a grounding in the methods and

17 procedures of science, while "knowledge" connotes more than

18 **subjective belief or unsupported speculation.** <u>Daubert I</u>, 509

19 U.S. at 589-90. The term "knowledge" "applies to any body of

20 known facts or to any body of ideas inferred from such facts or

21 accepted as truths on good grounds." <u>Id</u>. at 590 quoting

22 Webster's Third New International Dictionary 1252 (1986). In

23 order to qualify as "scientific knowledge," an inference or

24 assertion must be derived by the scientific method and proposed

25 testimony must be supported by appropriate **validation**- i.e. good

26 grounds, **based on what is known.** <u>Id</u>. The requirement that an

27 expert's testimony pertain to "scientific knowledge" establishes

28 **ORDER RE SUMMARY JUDGMENT-   648**

1 a standard of evidentiary reliability or "trustworthiness." Id.

2 and n. 9.

3   Klementiev's assumption (or hypothesis) that **one out of**

4 **every eight buckets of slugs** was transposed between May 1946 and

5 July 1947, and January 1949 and November 1949, amounts to no more

6 than subjective belief and unsupported speculation on his part.

7 Klementiev has not scientifically validated this assumption such

8 that it is reliable enough to warrant consideration by a trier of

9 fact.

10   Finally, even if Klementiev's assumption was somehow

11 validated, his failure to consider more than just the ten

12 dissolutions during Hanford's "start up" period renders his 1944-

13 49 release estimates inherently unreliable for the reasons

14 previously discussed.

15

16   c. **1950-60 Source Term Analysis**

17   (1) **HEDR Analysis**

18   For the period from 1950 to 1960, HEDR estimated a total of

19 42,802 Ci of I-131 was released. (Heeb 1994 at Table S.1, p.

20 vii). This is approximately 6% of the total release estimated by

21 HEDR for the period between 1944 and 1960 (738,773 Ci).[518]

22   HEDR did not conduct a (dissolver) batch-by-batch analysis

23 of cooling times for the period between 1950 and 1960, nor did it

24 model the day-to-day changes in reactor power levels and

25 ───────────────

26 [518] For the period between 1961 and 1972, HEDR estimated the
cumulative I-131 release at 226.5 Ci. (Heeb 1994 at Table S.1,
p. vii). Plaintiffs are not asserting any claims based on

27 exposure occurring solely after 1960.

28

ORDER RE SUMMARY JUDGMENT-   649

1 operations. Thus, for this period, HEDR did not attempt to

2 account for the decay of iodine during reactor shutdowns (as it

3 did for the 1944-49 period), but assumed all of the iodine which

4 could be created within the slugs was in fact created and present

5 at discharge (the "push").

6   According to Heeb 1994 at p. 4.15:

7   The ORIGEN2 burnup calculation which provided
             the curie per ton values is done at **constant
8   power** for the period of time sufficient to reach
             the burnup specified; that is the period of time
9   required for iodine-131 to reach a **steady-state
             (saturation) value** (Heeb 1993). Roughly 52 days
10   of steady operation are required to reach 99-percent
             iodine-131 saturation of the fuel, and the average
11   operating period actually ranged from 7 to 14 days.
             This shorter operating period meant that the fuel
12   was not actually saturated with iodine-131. There-
             fore, the use of ORIGEN2 to calculate curie per
13   ton values results in **overestimation** of the iodine
             -131 curie content at shutdown.

14 (Emphasis added).

15   If the reactor is run long enough, the I-131 reaches a

16 saturation value at which the amount of I-131 being produced is

17 the same as the amount of I-131 decaying. However, if the

18 reactor shuts down, the I-131 decays without I-131 being produced

19 and the I-131 in the fuel will not reach its peak saturation

20 value (99% within 50-55 day period). It will also not reach its

21 I-131 saturation value if the reactor has been shutdown in such a

22 way that the fuel does not reach saturation. (Klementiev 1995

23 Report at p. 11; Klementiev Dep. at pp. 252-55).

24   HEDR calculated a 15% "overestimate" from assuming all of

25 the iodine which could be created within the slugs was in fact

26

27

28 **ORDER RE SUMMARY JUDGMENT-   650**

1   created and present at discharge. (Heeb 1994 at p. 4.16[519];

2   Klementiev Dep. at p. 260). In other words, its iodine release

3   estimates for 1950-60 would have to be **decreased** by 15% based on

4   this "uncertainty" factor.

5   Instead of (dissolver) batch-by-batch analysis of cooling

6   times as had been done for the 1944-49 time period, HEDR used

7   **monthly average cooling times** in determining the amount of I-131

8   available for release during the 1950-60 time period.

9   Defendants note that HEDR's **Phase I** iodine release estimates

10   for 1944-47 were based on monthly average cooling times, as was

11   done by HEDR for the 1950-60 estimates. However, the **Phase II**

12   iodine release estimates for 1944-47 were based on specific

13   batch-by-batch cooling times, as has been discussed above

14   regarding Klementiev's analysis of the 1944-49 iodine release

15   estimates. (Heeb 1993 Vol. 1 at p. 5.1). Heeb reported the

16   results of the comparison between Phase I and Phase II estimates:

17   The average cooling time was shorter for the present
                 [Phase II] reconstruction based on a mass-averaged
18   cooling of **every dissolver cut** taken in time period;
                 59.6 days versus 61.7 [based on monthly average tons
19   dissolved and an average cooling time]. The difference
                 of 2.1 days amounts to a factor of 1.20 more iodine-
20   131.

21   (Id.)(Emphasis added). In other words, batch-by-batch cooling

22   time analysis yielded a 20% greater release of iodine (1.20 more

23   iodine) as compared to monthly average cooling time analysis for

24   **1944-47.**

25   For the 1950-60 time period, HEDR found the **uncertainty**

26   _____

     [519]   15% derived from 0.85 median and mean uncertainty
27   distribution for saturation. Table 4.5 of Heeb 1994.

28   **ORDER RE SUMMARY JUDGMENT-   651**

1   inherent in using monthly average cooling times, as opposed to

2   "actual fuel-batch cooling times within the month," produced a

3   14% "underestimate" in the amount of iodine released. (Heeb 1994

4   at p. 4.16).[520] In other words, HEDR's iodine release estimates

5   for 1950-60 would have to be **increased** by 14% based on this

6   "uncertainty" factor.

7   Finally, HEDR came up with a release factor to determine how

8   much of the I-131 gas actually was emitted from the stacks at the

9   separations plants (i.e. how much escaped the filters).[521]

10   (Heeb 1994 at pp. 4.10-4.13). It also analyzed the uncertainty

11   in the release factor. (Id. at pp. 4.16-4.17). HEDR's generic

12   release factor for the 1950 to 1960 time period is 1.25.

13

14   **(2)   Klementiev Analysis**

15   Klementiev takes issue with HEDR's use of monthly averaged

16   cooling times. Klementiev concludes the monthly averaged cooling

17   times as they appear in the historical records cannot be used by

18   HEDR for estimating I-131 releases because the "exponential

19   nature of I-131 decay is ignored in the averaging procedure."

20   According to Klementiev, when a proper averaging procedure is

21   employed, the monthly average cooling times decrease by 2-3 days

22   as compared to HEDR's values.   (Klementiev 1995 Report at p. 9).

23   _____

24   [520]   14% derived from 1.14 median and mean uncertainty
     distribution for cooling time. Table 4.5 of Heeb 1994.

25   [521]   T-Plant started up in December 1944 and shut down in
     February 1956. B-Plant started up in April 1945 and shut down in

26   June 1952. REDOX started up in January 1952. PUREX started up
     in January 1956. REDOX and PUREX were replacements for the B and

27   T Plants. (Heeb 1994 at p. 1.2).

28   **ORDER RE SUMMARY JUDGMENT-   652**

1 Less cooling time means more I-131.

2   Klementiev faults HEDR for not using the "minimum values of

3 the cooling times" available from the historical records.

4 According to him, if the minimum cooling times are taken into

5 account, HEDR estimates of I-131 releases must be increased by

6 10% to 30%. Id.

7   Klementiev contends that for the period after 1949, HEDR

8 fails to account for a bias in the monthly average cooling times.

9 According to Klementiev, on average HEDR's cooling times are

10 biased high by three days. Klementiev concludes that "if this

11 bias remained the same in the period after 1949, then all HEDR

12 estimates should be increased by about 20%." Id.

13   In addition to taking HEDR to task about its monthly cooling

14 times, Klementiev challenges HEDR's release factor. According to

15 Klementiev, because HEDR substituted the average (**mean**) value of

16 the release factor with the **median** value, "this led to an

17 underestimation of the releases." Id. Klementiev's generic

18 release factor is 4.5.

19   Furthermore, Klementiev asserts the operational data (stack

20 monitoring data) shows the release factor for some periods is

21 underestimated in the HEDR model and "[i]f this underestimation

22 is taken into account, the HEDR estimates should be increased by

23 more than 200%." For that proposition, Klementiev cites Dr.

24 Robert Jervis' 1995 report, "Evaluation of Radiochemical Aspects

25 of HEDR." Id. at p. 10.

26   Finally, Klementiev states that another source of

27 underestimation is that "[s]ome of the values of the cooling

28 **ORDER RE SUMMARY JUDGMENT-   653**

1 times were erroneously compiled from the historical records."

2 Id.

3   In his Scenarios 3 though 6, Klementiev takes these various

4 factors into account in arriving at I-131 release estimates.

5 Scenario 3 takes into account only what Klementiev refers to as

6 "Underestimation of Release Factor," arising from HEDR's use of a

7 median value instead of a mean value. It does not, however, take

8 into account findings by Dr. Jervis. Scenario 4 considers only

9 the cooling time factors: 1) accounting for exponential

10 character of decay; 2) utilizing the minimum values of the I-131

11 cooling time; 3) misinterpretation of the averaged values of the

12 cooling times; and 4) accounting for bias in the historical

13 records. Scenario 5 takes into account all of the cooling time

14 factors and Klementiev's release factor, but does not consider

15 Jervis' findings regarding release factor. Scenario 6, however,

16 considers all of the cooling time factors, Klementiev's release

17 factor, and Jervis' findings regarding release factor. (Id. at

18 pp. 97-98).

19   Table 7.7 at p. 113 of Klementiev's 1995 report shows the

20 total estimate for each of the scenarios: Scenario 3- 70,127

21 Ci; Scenario 4- 69,111 Ci; Scenario 5- 112,431 Ci; and Scenario

22 6- 247,349 Ci.

23

24   **(3) Reliability of Klementiev's Analysis**

25   **(a) Saturation/Reactor Bias Factor**

26   Defendants contend Klementiev's cooling time analysis is

27 scientifically unreliable because he ignored primary data and was

28
ORDER RE SUMMARY JUDGMENT-   654

1 selective about the data upon which he chose to rely.

2   First, defendants say that while Klementiev increased his

3 iodine release estimates because of HEDR's use of monthly average

4 cooling times, he unjustifiably ignored the need to lower his

5 estimates because of the saturation or "reactor bias" factor.

6 HEDR concluded that it "overestimated" the release of iodine by

7 15% because of its assumption that all of the iodine which could

8 be created within the slugs was in fact created and present at

9 discharge from the reactor. By HEDR's calculations, the 15%

10 "overestimate" from the "reactor bias" factor offsets the 14%

11 "underestimate" caused by use of monthly average cooling times.

12 In other words, it is a "wash."

13   At his deposition, Klementiev stated it was his belief that

14 HEDR had used the "saturation" or "reactor bias" figure in its

15 estimation procedure and had it not done so, its release

16 estimates would overestimate the iodine released by 15%.

17 (Klementiev Dep. at p. 260). In his affidavit, Klementiev says

18 he was aware of this issue, but did not mention it because it is

19 "absolutely irrelevant to the accounting for arithmetic averaging

20 of individual cooling times." (Klementiev 1997 Affidavit at p.

21 12).[522]

22   In their brief, plaintiffs' counsel likewise contend the

23 _____

24   [522] Elsewhere in his affidavit, Klementiev asserts that
defense counsels' brief initially says the saturation or "reactor
25 bias" factor was not considered, but then later states it was
considered. (Klementiev 1997 Affidavit at pp. 8-9). The court
26 has not found such an inconsistency in counsels' brief. The
statement quoted from Heeb 1994 at p. 4.15 makes it clear the
27 "reactor bias" factor was considered.

28 **ORDER RE SUMMARY JUDGMENT-   655**

1   "reactor bias" or "saturation" factor is irrelevant to

2   Klementiev's mathematical analysis of cooling times. However,

3   counsel go on to contend that HEDR minimized the "saturation"

4   effect, citing passages from Heeb 1993 Vol. 1 and a Hanford

5   historical document. They also contend that defendants

6   "conspicuously" leave out "peaking factor"[523] which plaintiffs

7   apparently argue would reduce or eliminate the offset between

8   cooling time and reactor bias. Plaintiffs' counsel contend there

9   is no offset between cooling time and reactor bias and that

10   "ignoring the combined effects of saturation, batch distribution

11   [aka "cooling time,"] and peaking factors, results in at least a

12   10% underestimate of HEDR's nominal release estimates."[524]

---

[523] "Peaking factor" is one of the factors considered in
HEDR's uncertainty analysis of the curies processed between 1950
and 1972. "Peaking factor" is the ratio of the average power of
fuel in a discharge to the average power of the reactor. Heeb
1994 at p. 4.15. HEDR reported the "mean" uncertainty as 1.033
(3%) and the "median" uncertainty as 1.04 (4%). Id. at p. 4.16.

[524] What plaintiffs mean by "nominal" estimates is that the
monthly estimates of the amount processed at each of the
separations plants from 1950 to 1972 as listed in Heeb 1994 at
Appendix B, Table B.1, do **not** incorporate the uncertainty
factors- saturation, peaking factor, batch distribution (cooling
time)- discussed in Heeb 1994, Table 4.5 at p. 4.16. Heeb
confirmed this in a letter to plaintiffs' counsel (Foulds Ex.
52), stating that "[t]o calculate the upper and lower bounds of
the uncertainty range for curies 'processed,' one must . . .
multiply the nominal values in Table [B.1] by the factors
presented in Table 4.5." The amount of curies "processed," of
course, bears on the amount of curies released, the amount
released being a fraction of the total amount processed.
      Plaintiffs' counsel point out that the "nominal" values in
Table B.1, without incorporation of the uncertainty factors,
appear to be the same values used in HEDR's "Atmospheric
Dosimetry Report." Farris 1994, Table B.1 at p. B.4. In other
words, in calculating doses for 1950-72, HEDR did not consider
the uncertainty regarding saturation, peak factor, and cooling
time.
      Defendants' counsel do not respond to this particular point.

ORDER RE SUMMARY JUDGMENT-   656

1 (Plaintiffs' Response Br. at pp. 38-39).

2   Plaintiffs' counsel do not attribute any of these arguments

3 to Klementiev. There is no citation to his report or to his

4 deposition. Klementiev's affidavit does not offer a substantive

5 reason why there should not be an offset between cooling time and

6 "reactor bias." Rather, Klementiev asserts "reactor bias" is

7 wholly irrelevant to this mathematical model. Hence, that is the

8 question: is it in fact irrelevant?

9

10   **(b) Monthly Average Cooling Times**

11   Klementiev states his mathematical model is necessary

12 because HEDR's monthly average cooling times are the result of

13 **arithmetic** averaging which ignores the "effect of **exponential**

14 decay." Klementiev says arithmetic averaging is okay so long as

15 special correction factors are developed to account for

16 exponential iodine decay and minimum cooling times. According to

17 Klementiev, those correction factors are computed by the use of

18 the mathematical model offered in his report. (Klementiev 1997

19 _____

20 It is not clear why HEDR apparently failed to consider the
   uncertainty, although perhaps HEDR determined it was
21 inconsequential because of the "offset" between the factors and
   the relatively small amount of I-131 it found was released
22 between 1950 and 1972. Nonetheless, even if HEDR did not
   incorporate the uncertainty into its release estimates for 1950-
23 72, that does not necessarily make Klementiev's analysis any more
   scientifically reliable.
24   Although HEDR may have ignored the results of its
   uncertainty analysis in its final release estimates, the fact is
25 that it conducted an uncertainty analysis which considered
   saturation, peak factor and cooling time. As will be discussed,
26 plaintiffs' counsel and Klementiev acknowledge that "uncertainty"
   analysis is necessary where monthly average cooling temperatures
27 are used.
28
   ORDER RE SUMMARY JUDGMENT-   657

1   Affidavit at p. 10). Essentially, Klementiev asserts he improved

2   the averaging procedure. Based on his averaging procedure,

3   Klementiev concludes HEDR underestimates iodine releases by about

4   60%. (Klementiev 1995 Report at p. 99).

5   The defendants do not attack Klementiev's averaging

6   procedure **itself**. They do not attack the accuracy of

7   Klementiev's mathematical equations. They seemingly do not

8   dispute Klementiev's assertion that HEDR committed an error by

9   not accounting in its averaging procedure for exponential decay

10 and minimum cooling times. What defendants do attack is

11 Klementiev's failure to review the actual historical batch data.

12 Defendants say Klementiev's mathematical model is only a

13 "hypothetical" approach. Defendants turn to plaintiffs' expert,

14 Dr. Thomas Cochran, as support for their argument that Klementiev

15 should have made an effort to validate his mathematical model by

16 a review of historical batch data.

17   In his revised report of March 19, 1996, "Errors in the

18 Source Term of the Hanford Environmental Dose Reconstruction,"

19 Cochran offered a critique of HEDR's monthly release estimates

20 for 1950-72. He concluded that in calculating monthly releases,

21 HEDR used arithmetic mean cooling times (monthly average cooling

22 times) which had the effect of underestimating releases.

23 (Cochran March 19, 1996 Report at p. 5). Using a **mathematical**

24 **formula**, Cochran calculated what he considered the "error

25 introduced by using the arithmetic average cooling time as a

26 function of the range of cooling times." Figure 1 at p. 27 of

27 Cochran's report shows the percent increase in release estimates

28 **ORDER RE SUMMARY JUDGMENT-   658**

1 required for a range of cooling times from 0 to 60 days. Cochran

2 shows a bias of up to 60% depending on the standard deviation

3 employed. This is similar to the 60% underestimate calculated by

4 Klementiev through his mathematical model.

5   Cochran went on in his report to consider **actual historical**

6 **cooling time data** pertaining to separations plant runs during

7 1950 to 1953, specifically: J.H. Wolff, "Dissolving Data- 271-B-

8 Building," (HW-4683-T)(Dec. 21, 1951); J.H. Wolff, "Dissolving

9 Data- 271-T Building," (HW-4684-T)(Dec. 21, 1951); and J.H.

10 Wolff, "Dissolving Data- S Plant," (HW-4685-T)(Dec. 21, 1951).

11 These are otherwise collectively known as the "Wolff" data.[525]

12 Cochran used this data to "estimate the bias introduced by

13 assuming average monthly cooling times during this 1950-1953

14 period."

15   His conclusion was that HEDR's estimate of I-131 released

16 should be increased by about 16% for 1950-51, by about 20% for

17 1952, and by about 30% in 1953 "to account for HEDR's failure to

18 properly treat the cooling times." According to Cochran, because

19 in subsequent years (1954-72) the average cooling times and the

20 range of cooling times increases, the bias would be even greater

21 for those years. Id. at p. 7. Overall, Cochran concluded that

22 HEDR's estimate of the **total** I-131 released between 1950-72

23 should be increased by about 20%, "due to the average cooling

24 time assumption alone." Id. at p. 7 and Table 9 at p. 26.[526]

---

25   [525] Defendants' Exs. 126, 127 and 128.

26   [526] At Table 9, Cochran proposes a 17% increase in HEDR's

27 total release estimate for 1950-72, from 43,038 Ci to 50,889 Ci.

28 **ORDER RE SUMMARY JUDGMENT-   659**

1 Defendants say Cochran's conclusion is consistent with the "14

2 percent-20 percent" bias range calculated by HEDR.[527]

3   Cochran was asked about Klementiev's mathematical approach

4 and the proper way for dealing with the 1950-72 cooling times.

5 His response:

6   Well, I think there are a variety of techniques
           that can be utilized, and some are more accurate,
7   than others, and it is not to say that any of
           these approaches taken by the other experts are
8   wrong.

9   **If you are asked to make an estimate, and you
           only have a few minutes, you might do a mathematical**
10   **approach.** If you have the time and the data, you
           can go back and do something more accurate, provided
11   the data is accurate and so forth.

12   . . . I tried to, as best as I could, to sort of
           scope it out with a mathematical assumption about
13   what the distribution of the cooling times might
           be over a monthly period, and then, where I had
14   access to the data, I tried to do it more care-
           fully. **And obviously, if you have the data, and**
15   **you trust the data, that technique should give**
           you the better answer.
16
           That's not to say the other techniques are wrong.
17   Different people will approach it slightly
           differently, depending on what data is available
18   and how much time they want to spend on that part
           of the problem.
19
   (Cochran Dep. at pp. 117-18)(Emphasis added).
20
       Klementiev acknowledged having access to each of the
21
   historical documents Cochran used (HE-4030; HW-4683-T; HW-4684;
22
   HW-4685-T) to estimate the bias introduced by use of average
23
   monthly cooling times.   (Klementiev Dep. at pp. 235-45).
24
   _____
25   [527] The 20% comes from HEDR's Phase I calculation of I-131
   release estimates for 1944-47 through use of monthly average
26 cooling times. The 14% is the uncertainty calculated by HEDR for
   1950-72 estimates because of the use of monthly average cooling
27 times.
28
   ORDER RE SUMMARY JUDGMENT-   660

1 Klementiev acknowledged he did not incorporate these batch-by-

2 batch cooling times into his analysis. He stated this was

3 because he wanted to compare his estimates with HEDR's estimates

4 and "since they used **monthly data only** I decided also to use

5 monthly data to compare apples and apples . . . ." (<u>Id</u>. at pp.

6 237-38)(Emphasis added).[528]

7   Klementiev admitted that "[i]t would be better for me" to

8 see the effect of average cooling time by modeling it on a batch-

9 by-batch basis using the historical data. Klementiev testified

10 he did not use the batch-by-batch data for 1950-72 for the same

11 reason he believed HEDR did not use it: "After '50 the releases

12 were pretty small so we could simplify our technique of

13 estimation and use still adequate but simplified technique on a

14 monthly basis." (<u>Id</u>. at p. 239).

15   Asked why he was making that same simplification when his

16 report appeared to be criticizing HEDR for doing it (using

17 monthly average cooling times), Klementiev responded:

18   I did this in the way how can I improve HEDR
            model, and actually it can be considered as
19   improvement of the HEDR model where more factors
            like minimum cooling time they ignored can improve
20   their calculations, so this is the way. **On
            the other hand, if I were given more time I**
21   **would definitely do what you said. I would
            definitely try to run my model MERI to check how**
22   **accounting for the detailed data influenced the
            result, but I was in the situation where my time**
23   **was too short.**

24 _____

25   [528] The "monthly data" referred to is S.P. Gydesen,
    "Selected Monthly Operating Data for B and T Plants, Redox and
26 Purex 1944-1972," (HW-89085)(April 1992). Hereinafter, it is
    referred to as "Gydesen 1992" or as the "monthly reports."
27 Foulds Ex. 37.
28
    ORDER RE SUMMARY JUDGMENT-   661

1 (Id. at p. 240)(Emphasis added). Klementiev acknowledged that in

2 using actual batch-by-batch cooling times, one would find the

3 **minimum value** of the cooling times of the batches, provided a

4 complete set of data was available. (Id. at p. 241).

5   Effectively, Klementiev attempts to correct the bias

6 inherent in HEDR's use of monthly average cooling times by

7 "improving" HEDR's "averaging procedure," without reviewing

8 batch-by-batch historical data. Essentially, he tries to correct

9 HEDR's math. For that reason, Klementiev asserts HEDR's **batch**

10 **distribution uncertainty analysis** involving saturation factor,

11 peaking factor, cooling time adjustment, etc., is **irrelevant** to

12 his mathematical analysis. He says his mathematical analysis is

13 a distinct analysis. According to Klementiev, he does not need

14 to look any further than the monthly data used by HEDR.

15   It appears Klementiev attempts to avoid a comparison between

16 the results of his mathematical model and the results from a

17 review of the historical data. In effect, he asks the court to

18 look just at the mathematical model itself- his equations and

19 calculations- and nothing else. As noted, the defendants do not

20 attack Klementiev's **equations and calculations.** The defendants

21 contend it is necessary to question at the very outset whether a

22 mathematical approach alone is the most scientifically reliable

23 way for assessing the bias inherent in using monthly average

24 cooling times.

25   In his affidavit, Klementiev makes various statements

26 showing his sole concern is with the mathematical model itself:

27   . . . the only way to argue against the

28 **ORDER RE SUMMARY JUDGMENT-   662**

1   mathematical model (or, to question the
          mathematical model validity) is to specify
2   the model's assumptions which the opponent
          does not agree with, or to specify the wrong
3   logic of the model formulation, or to show
          what was wrong with the mathematical trans-
4   formations.

5 (Klementiev 1997 Affidavit at p. 10).

6   He rebuts defendants' criticisms of his model by arguing

7 those criticisms are irrelevant to his "mathematical statement."

8 (Id. at p. 11). He asserts that HEDR's **uncertainty** explanation

9 has **"nothing** to do with the arithmetic averaging of individual

10 cooling times" and "nothing to do with the usage of minimum

11 cooling times." (Id.)(Emphasis in text). He adds, however, that

12 HEDR's calculation of uncertainty regarding cooling times (14%

13 underestimate) is mathematically correct even if it is

14 "irrelevant to the cooling time arithmetic averaging issue

15 considered in [his] report." (Id.) According to Klementiev:

16   The issue of uncertainty which was addressed by
          HEDR, is totally different from their arithmetic
17   averaging of an exponential decay factor,
          which is a question of incorrect mathematical
18   **procedure.**

19 (Id. at pp. 11-12)(Emphasis added).

20   Klementiev's entire focus is on the math, without regard to

21 the question of whether, as a methodologically sound practice, he

22 should have reviewed batch-by-batch historical data available to

23 him regarding the period from 1950 onward. Indeed, Klementiev

24 states he does not question the **pertinence** of Cochran's statement

25 that his (Cochran's) review of the historical data provides a

26 "better picture" of the effect of using monthly average cooling

27 times. However, Klementiev falls back on his standard refrain:

28 **ORDER RE SUMMARY JUDGMENT-**   **663**

1 "It has nothing to do with mathematics." (Id. at p. 14).

2 Loosely translated, this appears to be the equivalent of

3 Klementiev saying that if his math is okay, nothing else matters.

4   A question may arise that if it is not methodologically

5 sound for Klementiev to ignore batch-by-batch historical data,

6 what does that say about HEDR and its calculation of monthly

7 average cooling times based on monthly data, rather than batch-

8 by-batch data? Initially, the court notes it has never held that

9 HEDR is unassailable good science or that it is the default

10 standard. This motion in limine is not evaluated in that

11 context. This motion focuses on **Klementiev's** methodology.

12   HEDR specifically recognized the need "[t]o estimate the

13 **uncertainty** in curie content of iodine-131 due to the

14 distribution of **actual fuel-batch cooling time within the month**."

15 (Heeb 1994 at p. 4.16)(Emphasis added). HEDR recognized that if

16 it was not going to look at batch-by-batch historical data, it

17 needed to account for the uncertainty from not doing so.[529]

18   In their brief, plaintiffs' counsel say that while

19 Klementiev's **method of averaging cooling times** is totally

20 distinct from HEDR's **batch distribution uncertainty analysis**, the

21 uncertainty analysis "would have been done regardless of whether

22 it used exponentially or arithmetically averaged cooling times."

23 (Plaintiffs' Response Br. at p. 37). In other words, counsel

24 concede it is necessary to do an uncertainty analysis of cooling

25 time whether Klementiev's exponential averaging or HEDR's

26 _____

     [529] This is so, even if it apparently did not incorporate
27 that uncertainty into its final release estimates.
28
     ORDER RE SUMMARY JUDGMENT-   664

1 arithmetic averaging is used. However, there is no indication

2 from Klementiev or plaintiffs' counsel whether this uncertainty

3 analysis would be the same using Klementiev's model instead of

4 HEDR's model, or that it would provide the same results (14%

5 underestimate because of cooling time uncertainty; 15%

6 overestimate because of uncertainty regarding saturation factor).

7 As noted above, Klementiev utterly detaches himself from any

8 analysis accounting for uncertainty in saturation factor and

9 cooling times. One questions whether there would be the same

10 offset here because of the respective uncertainties regarding

11 saturation factor and cooling times.

12 Klementiev does not at all consider the impact of the

13 historical batch-by-batch data, whether that is through actual

14 consideration of such data, or through an uncertainty analysis.

15 Klementiev's 60% underestimate is based only on his exponential

16 averaging procedure. It is purely the result of mathematical

17 formulas applied to **monthly data**, the same monthly data used by

18 HEDR for its arithmetic averaging procedure.

19 Cochran's opinion that HEDR's use of monthly averaging

20 cooling times produces a 17% underestimate in I-131 releases is

21 arguably persuasive evidence corroborating the results of HEDR's

22 batch distribution uncertainty analysis as to cooling time.

23 However, it does not necessarily mean HEDR's analysis is correct.

24 Nor does the court need to make such a finding as part of its

25 Daubert analysis.

26 Plaintiffs argue it is no more than a random numerical

27 coincidence that Cochran's figures approximate HEDR's figures.

28 **ORDER RE SUMMARY JUDGMENT-   665**

1 They emphasize Cochran's statement that the bias would be even

2 greater than 30% in years subsequent to 1953. Plaintiffs contend

3 Klementiev hesitated to use the Wolff batch-by-batch data which

4 Cochran used in arriving at his 17% underestimate. Plaintiffs

5 say this is so because even HEDR itself cautioned that this data

6 was "not complete in all respects." S.P. Gydesen, "Documents

7 Containing Operating Data for Hanford Separations Processes,

8 1944-1972," (September 1992)(PNWD-2028 HEDR) (hereinafter,

9 "Gydesen 1992b").[530] According to plaintiffs' counsel,

10 Klementiev did not have at the time of his 1995 report the "Metal

11 Histories" against which to confirm whether "complete" dissolving

12 data for the applicable time periods were included in the Wolff

13 notebooks, and therefore he relied on HEDR's monthly data which

14 provided minimum cooling times for 1952 through 1960.[531]

15   In his affidavit, Klementiev asserts "the only historical

16 data other than the contractor's reports of monthly cooling

17 time[] averages was the Wolf (sic) processing data, which was not

18 complete enough to utilize for this analysis, nor did it cover

19 all the years in question, nor did HEDR try to use it."

20 (Klementiev 1997 Affidavit at p. 15). Klementiev is obviously

21 correct that HEDR did not use the batch-by-batch data in

22 calculating estimates for 1950-72. Furthermore, it is true that

23 the Wolff data covers only the period 1952-55. (Gydesen 1992(b)

---

[530] Foulds Ex. 36. This document should not be confused
with the document from which HEDR's "monthly data" is derived and
which is referred to herein as "Gydesen 1992."

[531] As it turns out, however, Klementiev was willing to rely
on the Wolff data for his release factor. See discussion _infra_.

ORDER RE SUMMARY JUDGMENT-   666

1   at pp. 3.4-3.5).

2   However, the fact there may be limitations in using the

3   Wolff data does not necessarily excuse consideration of it as

4   part of a methodologically sound analysis of the accuracy of

5   monthly average cooling times. Secondly, neither plaintiffs'

6   counsel or Klementiev say where in either his 1995 report or his

7   deposition he offered this as a reason for not reviewing the

8   **Wolff data. At his deposition, Klementiev stated he used the**

9   average and minimum cooling times available from the **monthly data**

10   (Gydesen 1992) and did not check to see if they were correct or

11   not. He just took the monthly data as is, which he claims is

12   what HEDR did. (Klementiev Dep. at p. 243).

13   In his 1995 report, Klementiev stated it was "reasonable to

14   suggest" that for the period after 1949, dates of "extraction"

15   were erroneously interpreted by HEDR **(in its monthly data-Gydesen**

16   **1992)** as dates of "dissolution." (Klementiev 1995 Report at p.

17   19). "Extraction" is a step which occurs **after** dissolution of

18   the uranium slugs in nitric acid. Consequently, if the

19   extraction date is erroneously interpreted as the dissolution

20   date, this results in inaccurate cooling times. The cooling

21   times will be inaccurately **long**, resulting in an underestimate of

22   the amount of I-131 released at the critical dissolution step.

23   It is at the dissolution step that the I-131 gas is released. In

24   other words, Klementiev suggests the monthly average cooling

25   times are actually shorter than HEDR reported in Gydesen 1992,

26   meaning in turn that more I-131 was released than estimated by

27   HEDR.

28
ORDER RE SUMMARY JUDGMENT-   667

1   According to Klementiev, the average difference between

2 monthly average cooling times as indicated in HEDR's monthly data

3 (Gydesen 1992) and the monthly average cooling time found in Heeb

4 1993 Vol. 2 equals 3.71 days. Put another way, the monthly

5 average cooling times in Gydesen 1992 are on average 3.71 days

6 more than those found in Heeb 1993 Vol. 2. Klementiev says

7 HEDR's misinterpretation of extraction dates as dissolving dates

8 causes about a 25 to 30% underestimate of the releases from 1950

9 onward. (Klementiev 1995 Report at p. 20).

10   Klementiev presents a chart for a period from January 1947

11 to September 1947 to illustrate his findings. (Klementiev 1995

12 Report at p. 20). Column D is the monthly average cooling time

13 from reactor "push" to dissolution found in Heeb 1993 Vol. 2.

14 Column E is the monthly average cooling time from "reactor" push

15 to dissolution **as reported** in Gydesen 1992. One can see that

16 each of the figures in Column E are higher than those in Column

17 D. Klementiev found the figures in Column E (Gydesen 1992) were

18 on average 3.71 days longer than the figures in Column D (Heeb

19 1993 Vol. 2). Column F contains figures derived from FTS-(XX)-

20 71, "Metal History and Percent Record," November 19, 1946.[532]

21 Those figures represent the monthly average cooling time in days

22 from reactor "push" to **extraction**. Klementiev points out the

23 similarity between the figures in Column F and Column E as

24 evidence that the monthly average cooling time as reported in

25 Gydesen 1992 is actually based on the number of days between

26 ──────────────

27   [532] Foulds Ex. 33.

28 **ORDER RE SUMMARY JUDGMENT-   668**

1   "push" and extraction, rather than "push" and dissolution.[533]

2   In their response brief, plaintiffs' counsel mention

3   Klementiev's findings and assert "production documents establish

4   that the underestimation is greater than **previously**

5   contemplated." (Plaintiffs' Response Br. at p. 29)(Emphasis

6   added). What follows for the next seven pages of the brief (pp.

7   29-36) is an analysis by **plaintiffs' counsel** in which there is no

8   citation to Klementiev's expert report. Counsel represent **their**

9   analysis is an extension of the findings made by Klementiev in

10 his 1995 report regarding misinterpretation of extraction times

11 as dissolving times.

12   Plaintiffs point out that in calculating I-131 release

13 estimates, HEDR used a "lag" factor for the August 1946 to

14 December 1947 time period where the principal references were to

15 metal history reports which did not give the day of dissolution,

16 but the day of extraction. (Heeb 1993 Vol. 1 at p. 4.23). The

17 "lag" time represents the difference between dissolution and

18 extraction. HEDR figured an average "lag" time of 3 days between

19 dissolution and extraction. (Id. at p. 4.24). Thus, HEDR looked

20 at the date of extraction and assumed that a date three days

21 earlier was the date of dissolution. Essentially, this is how

22 HEDR went about making their best determination of the actual

23 _____

24   [533] Column C of the chart is the average number of days from
    "push" to **shipping** as derived from the "200 North Area Shipment"
25 records, (HAN-45801). Those figures are consistently lower than
    the figures found in Column D representing average number of days
26 between "push" and dissolution. Plaintiffs argue that "the
    shipping date is effectively the initial dissolving date," but
27 these figures may suggest otherwise.

28
    ORDER RE SUMMARY JUDGMENT-   669

1 dissolution date for August 1946 to December 1947 period. As

2 noted, HEDR figured cooling time based on the difference between

3 "push" from the reactor and dissolution.[534]

4   **Plaintiffs' counsel** assert the value assigned by HEDR to the

5 "lag" between dissolving and extraction (3 days) is too low as

6 proven by the arithmetically averaged "lag" times which they

7 (counsel) calculated based on "200 Area Weekly Production

8 Reports." (Plaintiffs' Response Br. at pp. 30-31). According to

9 counsel, the "lag" times calculated by **them** translate to "more

10 than a 5% increase in releases from T and B Plants for the August

11 28, 1946 through December 31, 1947 period (when accounting for

12 the non-linear effect of exponential decay)." (Plaintiffs'

13 Response Br. at p. 31). In other words, if the "lag" time is

14 increased, that means the dissolution date is pushed back

15 earlier, decreasing the average time between the reactor "push"

16 and dissolution. The cooling time is shortened, decay is

17 lessened, and more iodine is released.

18   This is a new analysis which, by the admission of

19 plaintiffs' counsel, is mentioned nowhere in Klementiev's 1995

20 report. Klementiev does not discuss problems with HEDR's "lag"

21 value (3 days) and the purported consequences regarding

22 exponential decay. Furthermore, Klementiev specifically opined

23 that HEDR's alleged misinterpretation of extraction dates as

24 dissolving dates was irrelevant for the years **1944 to 1949**

25 _____

26   [534] It appears a three day "lag" factor would almost
   completely take care of the average 3.71 day discrepancy reported
27 by Klementiev for January 1947 to September 1947.
28

ORDER RE SUMMARY JUDGMENT-   670

1 because "[f]or this period information about timing of processing

2 of each particular cut was available from the historical records

3 and it was used in the HEDR Project." (Klementiev 1995 Report at

4 pp. 19-20)(Emphasis added).

5   Plaintiffs' counsel go on to review the FTS-179 and FTS-311

6 "Metal Histories."[535] According to counsel, HEDR's three day

7 "lag" period was also applied to these metal histories because

8 they provide dates of extraction rather than dissolution.

9 Counsel asserts the three day "lag" factor is wrongly applied

10 here as well.

11   Klementiev did **not** review these particular metal histories

12 in his 1995 report. From these metal histories, plaintiffs'

13 counsel calculate arithmetically averaged monthly "extraction"

14 cooling times (average number of days from "push" to extraction).

15 They also calculate arithmetically averaged monthly "dissolving"

16 cooling times (average number of days from "push" to dissolving).

17 Those are found in Table 1 of plaintiffs' brief at pp. 33-35.

18 The table pertains to the months from January 1948 through

19 December 1950.

20   Counsel calculate "lag" times by comparing the monthly

21 average dissolution time with the monthly average extraction

22 time. For example, in January 1948, the monthly average

23 dissolution time for B-Plant was 91.61 days, while the monthly

24 average extraction time was 99.28 days. The difference between

25 these two figures, the "lag," is 7.67 days. According to

26 _____

27 [535] FTS-XX-179 is "Metal History and Percent Record," (July
   1, 1947). Foulds Ex. 32.

28 **ORDER RE SUMMARY JUDGMENT-   671**

counsel, Table 1 reveals the average monthly "lag" for B-Plant is 7.69 days and for T-Plant it is 6.12 days. If the "lag" time is increased, that means the dissolution occurred at an earlier time, lessening the cooling time and increasing the amount of I-131 released. **Counsel** conclude as follows:

> Because of the exponential decay of iodine, the actual effect of the difference to the HEDR estimates must be analyzed month-by-month, however, the overall difference would be at least 20%. The difference in HEDR is even greater for the post-49 time period for which no 'LAG' was applied. Applying the actual 'lag' times computed from the Metal Histories to the post-49 calculations for 'B' and 'T' Plants **means a nearly 44% underestimate by HEDR for these plants.**

(Plaintiffs' Response Br. at p. 35)(Emphasis in text).

This is nothing more than speculation on the part of plaintiffs' counsel. The court also notes that counsel's chart pertains to the period from January 1948 through 1950 and therefore, covers only one year of the relevant 1950-60 time period. For the years **1944 to 1949**, Klementiev states misinterpretation of extraction dates as dissolution dates is inconsequential because information about timing of the processing of each particular cut was available from the historical records.

Furthermore, plaintiffs must concede once again that this "lag" analysis is nowhere to be found in Klementiev's report. Indeed, plaintiffs' counsel argue "[t]hese are not calculations that require expert testimony or specialized scientific knowledge, but is information taken straight from the Metal Histories and the arithmetic averages computed on a desk calculator." While counsel may have the ability to do the simple

ORDER RE SUMMARY JUDGMENT-   672

1   math involved, an analysis of dissolving and extraction times and

2   most importantly, the conclusions derived therefrom, must be

3   presented by an expert. Defense counsel would then have an

4   opportunity to depose the expert about the analysis and

5   conclusions.

6   Plaintiffs' entire discussion of dissolution dates versus

7   extraction dates is a diversion from the critical issue: as a

8   matter of sound scientific methodology, should Klementiev have

9   considered the **actual batch-by-batch historical data** available

10 for the period after 1949? Had he done so, perhaps Klementiev

11 would not have needed to consider the alleged misinterpretation

12 of extraction dates as dissolving dates found in HEDR's **monthly**

13 **data**. Likewise, the "lag" time analysis offered by **plaintiffs'**

14 **counsel** ignores batch-by-batch data in favor of monthly averages.

15

16   **(c)   Release Factor**

17   The release factor is the ratio of I-131 released to the

18 radionuclide activity processed. HEDR calculated a monthly

19 release factor for each of the separations plants in operation.

20   Tables 7.2 through 7.5 at pp. 102-112 of Klementiev's 1995

21 report are release estimates for each of the separations plants

22 (T-Plant, January 1950 to February 1956; B-Plant, January 1950 to

23 June 1952; REDOX, January 1952 to December 1960; and PUREX,

24 January 1956 to December 1960). Under the heading "RF HEDR,"

25 Klementiev lists HEDR's monthly release factor. Thus, for

26 December 1951 at the T-Plant, the HEDR monthly release factor is

27 listed as 0.012. (Table 7.2 at p. 102). The amount released

28 **ORDER RE SUMMARY JUDGMENT-   673**

1 through application of HEDR's release factor is found under the

2 heading "Scnrio 1 Relsd HEDR," which stands for "Scenario 1

3 Released HEDR." HEDR's generic release factor is 1.25,

4 equivalent to a 98.75% median value for filter efficiency. This

5 release factor is derived from J.H. Warren, "Control of I-131

6 Releases to Atmosphere," (HW-68392) (1961), hereinafter "Warren

7 1961."[536]

8   For some months, Klementiev "adjusted" the release factor

9 upwards, meaning more I-131 was released from the amount of

10 material processed. Klementiev's "adjusted" release factors are

11 found in his Tables 7.2 through 7.5 under the heading "Adjusted

12 RF." Thus, for December 1951 at the T-Plant, Klementiev

13 increases HEDR's release factor to 0.045. (Table 7.2 at p.

14 102).[537] The amount released applying Klementiev's "adjusted"

15 release factor is found under the heading "Scnrio 3 'Release

16 Factor,'" which stands for "Scenario 3 Release Factor."

17 Klementiev's generic "adjusted" release factor is 4.5, 3.6 times

18 higher than HEDR's generic release factor. A release factor of

19 4.5 is equivalent to a 95.5% filter efficiency.

20   While HEDR concludes 42,802 Ci of I-131 was released from

21 all the separations plants between 1950 and 1960, Klementiev's

22 "adjusted" release factor produces a total of 70,127 Ci under his

23 ───────────────

[536] Foulds Ex. 116.

24
[537] For the T and B-Plants, HEDR's release factor and
25 Klementiev's "adjusted" release factor are the same until after
   May 1951. After May 1951, Klementiev's monthly "adjusted"
26 release factor is always higher than HEDR's release factor. For
   REDOX and PUREX, Klementiev's monthly "adjusted" release factor
27 is always higher than HEDR's release factor.
28
ORDER RE SUMMARY JUDGMENT-   674

1 Scenario 3.   Scenario 3 considers **only** Klementiev's "adjusted"

2 release factor.   (Table 7.7 at p. 113 of Klementiev 1995 Report).

3   Scenario 5 takes into account Klementiev's "adjusted"

4 release factor, along with his various cooling time factors

5 (accounting for exponential character of decay, utilizing the

6 minimum values of the I-131 cooling time, misinterpretation of

7 the averaged values of the cooling times, etc.).   Klementiev's

8 Scenario 5 estimate is 112,431 Ci.   (Table 7.7 at p. 113 of

9 Klementiev 1995 Report).

10   Scenario 6 is the same as Scenario 5, **except** that Klementiev

11 adds a 2.2 release factor found in Dr. Robert Jervis' 1995

12 report, "Evaluation of Radiochemical Aspects of HEDR."   In

13 Klementiev's Tables 7.2 through 7.5, Jervis' release factor is

14 found under the heading "Jervis RF Scen 6."   Using the example of

15 December 1951 at the T-Plant, Jervis' release factor is 0.099, a

16 2.2 increase over Klementiev's "adjusted" release factor of 0.045

17 (0.045 x 2.2 = 0.099).   (See Klementiev Dep. at p. 235).

18   Section 6.1.1 of Klementiev's 1995 report is devoted to

19 "Release factor."   Klementiev says he examined measurements of

20 the I-131 release factors made at the REDOX plant from January

21 31, 1959 to December 31, 1959 available in Warren 1961.   Warren

22 reports the **median** value of release factor (MVRF) for 1959 at the

23 REDOX plant (0.0125)[538], and for 1959 and 1960 at the PUREX

24 plant (0.002).   (Klementiev 1995 Report at p. 84).

25   Klementiev asserts the use of these MVRFs in HEDR could be

26

27   [538]   From whence comes HEDR's generic release factor of 1.25.

28 **ORDER RE SUMMARY JUDGMENT-   675**

1 questioned for several reasons: 1) the MVRF was retrospectively

2 applied to REDOX operation prior to 1959, but evidence showed

3 that during the first few years of REDOX operation (and even

4 after 1960) the filter efficiency of the silver reactors[539] was

5 **not** as high as presented in Warren 1961; 2) the removal

6 efficiency figures offered in Warren 1961 and used by HEDR were

7 obtained for the complete Reactor-Absorber-Scrubber series, but

8 since this was not put into service until late 1957, the removal

9 efficiency figures could not be applied to any period prior to

10 late 1957;   3) the MVRF found for REDOX was applied to the T-

11 Plant and the B-Plant, an application which could be questioned

12 since it was not clear if the filtering systems of the T-Plant

13 and B-Plant in their first years of operation were as efficient

14 as the REDOX filtering system; and 4) Dr. McNeill questioned the

15 correctness of using **median** value instead of the **average (mean)**

16 value of the release factor for the estimation of monthly

17 averaged I-131 releases.[540]   (Id. at p. 85).

18   In his report, Klementiev concluded the removal efficiency

19 figures found in Warren 1961 were erroneous for separations plant

20 operations from 1950 to 1957. He therefore offered an

21 "alternative" set of release factor values based on "55

22 measurements" of release factors found in the "historical

23

24   [539] "Silver reactors" consist of columns packed with Beryl
saddles coated with silver-nitrate and designed to chemically
25 react and remove elemental iodine from dissolver off-gas lines.
The off-gas lines connect the dissolver to the stack for
26 discharge to the atmosphere. (Heeb 1994 at p. 4.10).

27   [540] McNeill November 1995 Report at p. 7. Foulds' Ex. 74.

28 **ORDER RE SUMMARY JUDGMENT-   676**

1 records." Klementiev found these measurements provided an

2 **average** value of release factor equal to 4.5, which was 3.6 times

3 higher than suggested by HEDR for the T-Plant, B-Plant, REDOX and

4 PUREX for the period from 1950 to 1957. (<u>Id</u>. at p. 95).

5   Defendants contend **Klementiev's** "adjusted release" factor is

6 without any evidentiary or documentary support. Defendants point

7 to a portion of Klementiev's deposition testimony wherein he was

8 unable to explain how he arrived at his "adjusted" release

9 factor. Asked whether his "adjusted" release factor represented

10 the **mean** value of the release factor estimates from Warren 1961

11 rather than the **median** value, Klementiev responded "probably,"

12 although he could not recall. He testified he would need to

13 review the records and check how he had calculated this

14 parameter. (Klementiev Dep. at pp. 233-34).

15   In his affidavit, Klementiev clarifies that the "55

16 measurements" referred to in his report are not the **number** of

17 measurements taken, but instead refers to when the measurements

18 were taken- **1955**. Klementiev says he inadvertently omitted an

19 apostrophe before "55." According to Klementiev, the 1955

20 measurements are the quarterly reported averaged measurements of

21 filter efficiency calculated for the REDOX filtering system and

22 found in J.H. Wolff, "Dissolving Data-S Plant" (HW-4685-T) (Dec.

23 21, 1951).[541] (Klementiev 1997 Affidavit at p. 16).

24   In his affidavit, Klementiev explains how he arrived at a

25 4.5 release factor:

_____

[541] Hereinafter, "Wolff 1951." Defendants' Ex. 128.

ORDER RE SUMMARY JUDGMENT-   677

> Two figures related to the first two quarters
> of 1955 are presented in [Wolff 1951]. For the
> first quarter of 1955, reported efficiency was
> 99%, and for the second quarter- it was 92%. No
> more data is available for the later period.
> Clearly, the average of 99% and 92% is 95.5%,
> which corresponds to a release factor of 4.5%
> . . . .
>
> The value of filter efficiency equal to 4.5% suggested
> as the input data for my model **is** the measured
> historical data.

(Klementiev 1997 Affidavit at pp. 16-17)(Emphasis in text).

In a footnote in his affidavit, Klementiev adds that 1955 was not the worst year for the S-Plant[542], noting the average filter efficiency reported for the third quarter of 1954 was 92.6%, and for the fourth quarter 91.2%. Therefore, Klementiev asserts he had "chosen pretty conservative (in favor of HEDR) measurements. (Id. at p. 16, n. 10).

Defendants contend that here again Klementiev ignores the whole of the available historical data and selectively chooses the removal efficiencies reported in Wolff 1951 for the first and second quarters of 1955. Defendants note the following about the Wolff REDOX filter efficiency data: 1) for the third quarter of 1954, the estimated total curies dissolved was 1200, with the silver reactor efficiency reported at 92.6% (Wolff 1951 at p. 50); 2) for the fourth quarter of 1954, the estimated total curies dissolved was 473, with a silver reactor efficiency reported at 91.2% (Id. at p. 54); 3) for the first quarter of 1955, the estimated total curies dissolved was **20,460**, with the silver reactor efficiency reported at **99%** (Id. at p. 57); and 4)

---

[542]   "S-Plant" is presumably another name for REDOX.

ORDER RE SUMMARY JUDGMENT-   678

1 for the second quarter of 1955, the estimated total curies

2 dissolved was 1106 (1105.84), with the silver reactor efficiency

3 reported at 92% (Id. at p. 60).

4   In addition to their contention that Klementiev is

5 unscientifically selective about his choice of data, defendants

6 contend Klementiev neglects to properly weigh the efficiencies

7 reported for the first and second quarters of 1955. The 92%

8 efficiency reported for the second quarter of 1955 involved 1106

9 curies, whereas the 99% efficiency reported for the first quarter

10 of 1955 involved 20 times that amount- 20,460 curies. Defendants

11 say a correct presentation of **all** the data (for all four

12 quarters), and a correct averaging of the data produces a filter

13 efficiency of 98.2%. This is close to the 98.75% median

14 efficiency value used by HEDR, and a significant increase over

15 Klementiev's 95.5% efficiency.[543] The court finds no reason to

16 quibble with these figures and it is perfectly logical to assign

17 greater proportional weight to the period during which the most

18 material was dissolved.

19   Defendants contend that had Klementiev reviewed the entire

20 set of stack data, he would have discovered the measured stack

21 emissions for 1952-55 was 3,415 curies. Defendants cite J.D.

22 Anderson, "Emitted and Decayed Values of Radionuclides in Gaseous

23 Wastes Discharged to the Atmosphere from the Separations

24 Facilities through Calendar Year 1972," (ARH-3026)(March 1,

25 _____

26   [543] A **98.2%** efficiency means that of the total 23,239 curies
dissolved during the four quarters, 424 curies of I-131 would

27 have been released to the atmosphere.

28 **ORDER RE SUMMARY JUDGMENT-**   **679**

1974).[544]   Table I of Anderson 1974 (p. 5), "Known Radioactivity

in Gaseous Waste Discharged from the Separation Facilities,"

indicates that from 1952 through 1955, 3,415 curies were released

(967 + 730 + 538 + 1,180 = 3,415).   According to defendants,

HEDR applied its 1.25 release factor (98.75%) to each of the

three operating separations plants (B-Plant, T-Plant and

REDOX[545]) for each of these four years (1952 through 1955).

Because HEDR calculated a total of 668,092 Ci was dissolved

during these years, defendants say a release of 3,415 Ci

translates into a 99.5% efficiency which is higher than the

98.75% median efficiency value employed by HEDR.

Plaintiffs assert Klementiev's "adjusted" release factor is

"reasonably reliable and scientifically supported because it is

based on a set of silver reactor efficiencies as reported by the

contractor [Wolff 1951]." However, they do not mention the other

quarterly data found in Wolff (Third and Fourth Quarter of 1954),

nor the fact the 99% efficiency reported by Wolff for First

Quarter 1955 involved 88% (20,460 Ci) of the estimated total

curies dissolved between Third Quarter 1954 and Second Quarter

1955.

A portion of Dr. McNeill's report is devoted to "Filtering."

He asserts that HEDR's use of a median filter efficiency value is

the wrong one to use, and that the average (mean) is appropriate

"to give a picture of the biological potential risk on the linear

---

[544]   Defendants' Ex. 164.

[545]   PUREX did not start operating until January 1956.

ORDER RE SUMMARY JUDGMENT–   680

1   hypothesis."   (McNeill November 1995 Report at p. 5).   He

2   concludes:

> [HEDR] underestimate[s] release fractions in the
> 1950s by using a 'generic release factor' based
> on median values in 1959 and 1960 rather than
> mean values.   This results in an underestimate
> of release in the 1950s of 45% for the B, T and
> REDOX plants and 25% for PUREX.

(Id. at p. 7).

While McNeill proposes a 145% (1.45) increase in HEDR's

release factor to account for HEDR's use of a median value, that

is still well below Klementiev's proposed 360% (3.6) increase in

the release factor.   Moreover, McNeill arrives at his figure by

way of a "linear hypothesis" that biological effect is

proportional to dose.   Klementiev gets his figure from the Wolff

data.   The court fails to see how McNeill serves as any support

for Klementiev's "adjusted" release factor.

This brings us to Dr. Jervis' findings in his 1995 report,

"Evaluation of the Radiochemical Aspects of HEDR."   Plaintiffs

say "Jervis' opinion that the contractor's reported [I-131]

emissions are systematically underestimated by 220% [a 2.2

release factor] . . . is **exclusive** of Klementiev's application of

an average release factor of 4.5 derived from **other** reported

silver efficiency data," referring to Wolff 1951, "Dissolving

Data-S Plant," (HW-4685-T).   If it is "exclusive," that appears

to mean the two release factors (Jervis' factor and Klementiev's

factor) are independent and can survive without one another.

Jervis assessed the validity of the I-131 release monitoring

(**stack sampling**) "by reviewing typical radioiodine measurements

ORDER RE SUMMARY JUDGMENT-   681

1 in stack gas samples . . . ." (Jervis 1995 Report at p. 4). He

2 identified several factors which "could have" affected the stack

3 measurements. Jervis opined that because of those factors,

4 releases could be underestimated by "as much as a factor of 2.2,

5 possibly higher in the 50's when measurements were sparse and

6 techniques crude." (Id. at p. 6). Among the factors identified

7 by Jervis were "plating out" in the long **sampling** lines (30 to

8 70% losses) and failure to account for decay between time of

9 **sample** collection and radiochemical analysis in the laboratory

10 (12-20% losses). Jervis concluded:

11   [I]t is probable in this opinion that the
           Hanford stack monitoring provided estimates
12   of I-131 releases that were from 75% [1.75]
           to 130% [2.3] low and some underestimates may
13   have even been lower in certain circumstances
           such as when high efficiency of the silver beds
14   increased the fraction of organic iodide released.

15 (Id. at p. 9)(Emphasis added).

16   Defendants argue Jervis' work provides no support for

17 Klementiev's release factor. Defendants say it is improper for

18 Klementiev to add Jervis' 2.2 release factor increase to his

19 [Klementiev's] proposed 3.6 release factor increase.[546]

20 According to defendants, this is borne out by a comparison of

21 HEDR's release estimate for each year from 1950 through 1960 with

22 the stack measurements for each of those years. The stack

23 measurements, of course, are based on historical data.

24   This comparison of release estimates and stack measurements

25 _____

[546] This makes for an approximate total increase of 5.8 (3.6
26 + 2.2) in the release factor. It is this increase which produces
   Klementiev's Scenario 6 estimate of 247,349 Ci (42,802 x 5.78).
27 This is also Klementiev's highest estimate.

28

ORDER RE SUMMARY JUDGMENT-   **682**

1 is illustrated in a chart prepared by defendants. (Defendants'

2 Reply Brief at p. 20). The stack measurements are taken from

3 Anderson 1974 at p. 2. The release estimates can be found in

4 Heeb 1994 at p. vii. The stack measurements found in the chart

5 for 1950 and 1951 (1,140 and 14,800 Ci respectively) are less

6 than the figures found in Anderson 1974 for 1950 and 1951 (2,140

7 and 18,700 Ci respectively). Defendants say this is because for

8 1950, stack measurement values include only the last four months

9 at the T-Plant due to the fact these are the only months for

10 which historical data is available. For 1951, stack measurement

11 values are only for the T-Plant because B-Plant data is not

12 available for the entire year.

13   The release estimates found in the chart for 1950 and 1951

14 (1,777 Ci and 16,100 Ci respectively) are less than the release

15 estimates found in Heeb 1994 for 1950 and 1951 (5,379 Ci and

16 27,397 Ci respectively). Defendants indicate that for comparison

17 purposes, the release estimate for 1950 contained in the chart

18 covers only the last four months at the T-Plant, like the stack

19 measurement figure. The release estimate for 1951 covers only

20 the T-Plant since it is only for the T-Plant that stack

21 measurement data is available for the full calendar year 1951.

22   Defendants' chart shows a cumulative release estimate of

23 27,903 Ci for 1950-1960 and a cumulative stack measurement

24 estimate of 21,161 Ci. Thus, HEDR estimates more I-131 released

25 than is shown by the stack measurement data.

26   Jervis states that Hanford stack measurements (stack

27 monitoring data) provide estimates of I-131 releases which are 75

28 **ORDER RE SUMMARY JUDGMENT-   683**

1 to 130% low. Therefore, according to defendants, Jervis' opinion

2 would result in an increase of the stack measurements in Anderson

3 1974 from 21,161 Ci to 37,032 Ci (21,161 x 1.75) or as high as

4 48,670 Ci (21,161 x 2.30). 37,032 Ci is a 33% increase over the

5 **HEDR release estimate** of 27,903 Ci for the 1950-60 period.

6 48,670 Ci is an approximate 75% increase over the **HEDR release**

7 **estimate.**

8   Defendants contend Klementiev improperly applies Jervis's

9 **stack sampling** correction to **HEDR's release estimates** because: 1)

10 Jervis did not base his figures on HEDR's release estimates, but

11 rather on the stack monitoring data; and 2) HEDR's release

12 estimates are already higher than the stack measurements. A

13 review of Jervis' report confirms Jervis' figures are based

14 wholly on the stack monitoring data. He does not consider the

15 HEDR **release estimates** for 1950-60, nor does he adjust those

16 estimates.

17   According to defendants, an even more important problem is

18 that while Jervis attempts to provide an **"independent**

19 comprehensive estimate of iodine emissions,"** Klementiev treats

20 Jervis' work as an **adjustment** to HEDR's release estimates. The

21 result, according to defendants, is that Klementiev improperly

22 adds Jervis' stack sampling adjustment on top of Klementiev's

23 cooling-time and filter-efficiency adjustments. In the process,

24 say defendants, Klementiev accounts three times for the same

25 alleged errors.

26   The stack is the **last stop** before I-131 reaches the

27 atmosphere. The I-131 has already gone beyond the filters (the

28 **ORDER RE SUMMARY JUDGMENT-   684**

1   silver reactors). Cooling time has already occurred, determining

2   the amount of decay and in turn, the amount of I-131 available

3   for release. Accordingly, Jervis' release factor, based on the

4   stack monitoring data, takes into account the silver reactor

5   efficiency which is the basis for Klementiev's release factor.

6   It is a matter of using **either** Klementiev's "adjusted"

7   release factor or Jervis' release factor, but not **both**. For

8   reasons set forth above (selectivity of data, calculational

9   error, etc.), Klementiev's "adjusted" release factor is

10   unreliable. Consequently, if any release factor is used, it

11   should be Jervis' release factor. The court notes there is no

12   indication of Jervis approving Klementiev's release factor.

13   Defendants do **not** attack **Jervis'** analysis on Daubert

14   grounds. They do not question the scientific reliability of

15   Jervis' release factor. The question then is whether Jervis'

16   release factor, by itself, is of any value to plaintiffs.

17   Although Jervis provides a fairly wide percentage range of how

18   far off he thinks HEDR's release estimates might be (75% to

19   130%), he does not, as far as the court can discern, provide what

20   he believes is a best estimate of the total I-131 release from

21   Hanford for 1950-60. The plaintiffs left that task to

22   Klementiev. For reasons set forth above, and to be summarized

23   subsequently, Klementiev's source term estimates for the 1944-49

24   and 1950-60 time periods are not scientifically reliable.

25   Plaintiffs discuss the efficiency of "water scrubbers" used

26   in an attempt to filter iodine from the dissolver off-gas lines.

27   Citing to various documents, **plaintiffs' counsel** conclude that

28   **ORDER RE SUMMARY JUDGMENT-   685**

1 water scrubber efficiencies may have been as low as 30% which

2 "would at least double HEDR's estimates for releases for the

3 period of May 1948 through December 1950." There is no

4 indication that Klementiev arrived at any conclusions about water

5 scrubber efficiencies and how they might impact release

6 estimates.[547]

7

8   (d)   **Summary**

9   Klementiev's source term analysis for the 1950-60 time

10 period is plagued by the same methodological deficiencies as his

11 1944-49 analysis, namely failure to consider all of the available

12 data and reaching conclusions which are without foundation in the

13 available data. Essentially, Klementiev attempts to reduce all

14 of this to a mathematical exercise, seemingly detached from what

15 took place in the plutonium production process.

16   The methodology behind Klementiev's 1950-60 source term is

17 unreliable where he failed to consider the impact of the reactor

18 bias/saturation factor and failed to consider batch-by-batch data

19 regarding the cooling time issue. While HEDR did not consider

20 batch-by-batch data in its 1950-60 source term analysis, it

21 recognized an uncertainty analysis was necessary to account for

22 the distribution of actual fuel-batch cooling time. The

23 plaintiffs emphasize Klementiev's modification of HEDR's

24 averaging procedure is distinct from HEDR's uncertainty analysis.

25 _____

26   [547] The analysis of **plaintiffs' counsel** pertains to May 1948
through December 1950, covering only one year of the relevant
27 1950-60 time period.

28 **ORDER RE SUMMARY JUDGMENT—   686**

1 That is true, but Klementiev fails to explain how the uncertainty

2 analysis affects the results of his modified averaging procedure

3 (i.e. what is the impact of an uncertainty analysis on the 60%

4 increase in release estimates suggested by Klementiev using an

5 exponential averaging procedure?). Factors in the uncertainty

6 analysis include both the **reactor/bias** saturation factor and the

7 cooling time factor. Under HEDR's uncertainty analysis, those

8 two factors almost completely offset each other.[548]

9   There is no question about the methodological deficiency in

10 Klementiev's release factor. This release factor is based on a

11 selective use of limited historical data. Besides the

12 unreliability inherent in his failure to consider **other** available

13 historical data, even the **limited data** selected by Klementiev

14 does not support his release factor. The release factor is

15 important. It is an integral component in figuring just how much

16 I-131 may have escaped through the stacks. Consequently, if

17 **Klementiev's** release factor is deficient, the rest of his

18 analysis regarding monthly average cooling time is of no value.

19   Defendants do not attack Jervis' release factor on <u>Daubert</u>

20 grounds. However, as noted, plaintiffs did not entrust Jervis to

21 come up with a best estimate of the amount of I-131 released.

22 That was Klementiev's responsibility. Jervis provides a

23 percentage range of the extent to which stack sampling

24 _____

25   [548] The court recognizes the possibility that faulting
    Klementiev for failing to consider batch-by-batch data **could**
26 somehow also point to a methodological deficiency in HEDR's
    analysis. However, the existence of a methodological deficiency
27 in HEDR would not make Klementiev's analysis any more reliable.

28 **ORDER RE SUMMARY JUDGMENT-   687**

1 inefficiency may impact release estimates, but Jervis does not

2 provide any actual release estimates.

3

4   **d.   Daubert Criteria**

5   Klementiev's I-131 source term analysis is **not** derived from

6 legitimate preexisting research unrelated to this litigation.

7 The record indicates Klementiev was specifically hired by

8 plaintiffs' counsel for the purpose of scrutinizing HEDR's iodine

9 release estimates. In his 1995 report, Klementiev states that

10 "[f]or the last two years I have worked on research and modeling

11 the Hanford releases of radioiodine" and acknowledges "[t]his

12 work has been done at the request of the Hanford Litigation

13 Office." (Klementiev 1995 Report at p. 4).

14   Klementiev does not dispute that his modeling of Hanford

15 radioiodine releases has **not** been subject to peer review. In

16 their response brief, plaintiffs' counsel attack the peer review

17 of the **HEDR** model as being "cursory." (Plaintiffs' Response Br.

18 at pp. 8-13). The alleged inadequacy of the **HEDR** peer review

19 does not change the fact that **Klementiev's** work has not been peer

20 reviewed.

21   There is nothing remotely suggesting Klementiev's work is

22 "generally accepted" within the scientific community. Indeed, as

23 noted above, even the two experts whose names Klementiev invokes,

24 McNeill and Jervis, do not endorse either his methods or his

25 conclusions. McNeill clearly qualifies his opinion about the

26 accuracy of Klementiev's release estimates, stating it all

27 depends on the accuracy of Klementiev's underlying assumptions.

28 **ORDER RE SUMMARY JUDGMENT-   688**

1 McNeill and Jervis do not validate Klementiev's methods or his

2 conclusions.

3   Considering these criteria in conjunction with the

4 methodological deficiencies discussed above (i.e. selective use

5 of data, data which provides no foundation for assumptions and

6 conclusions, etc.), Klementiev's iodine source term analysis

7 fails the "reliability" prong of <u>Daubert</u>.

8

9   **e. Fit/Relevancy**

10   Because <u>Daubert's</u> reliability prong is not satisfied, the

11 fit/relevancy prong need not be considered. However, in this

12 case, failure to satisfy the reliability prong also means the

13 fit/relevancy prong is not satisfied. Because the assumptions

14 underlying Klementiev's source term estimates are without

15 foundation, those estimates are so speculative they do not raise

16 an issue of material fact about the amount of Hanford iodine

17 emissions. Therefore, Klementiev's analysis cannot assist a jury

18 in determining a fact in issue.

19

20   **f. Qualifications**

21   Previously, with regard to Klementiev's "process analysis"

22 of plutonium emissions, it was pointed out that this litigation

23 represents Klementiev's first foray into radionuclide source term

24 estimation and that he has never conducted original scientific

25 research into how radionuclides are released from any type of

26 manufacturing process.

27   Plaintiffs emphasize the contributions of Drs. McNeill (a

28
ORDER RE SUMMARY JUDGMENT-   689

1 nuclear physicist) and Jervis (a radiochemist), apparently

2 suggesting that whatever Klementiev may lack in the way of

3 qualifications is remedied by McNeill and Jervis. Jervis says

4 nothing about Klementiev's methods or conclusions. As noted

5 above, McNeill qualifies his endorsement of Klementiev's

6 **conclusions (release estimates)**. McNeill expressed reservations

7 about the **method** by which Klementiev arrived at those

8 conclusions.

9   Plaintiffs note that Klementiev has a Ph.D. in applied

10 mathematics which includes an emphasis in computer modeling and

11 dynamic systems. In 1991, Klementiev received a second Ph.D. in

12 "mathematical epidemiology" in connection with his work at

13 Chernobyl.[549] Plaintiffs and Klementiev emphasize his prior

14 experience in developing mathematical and computer models.

15 (Klementiev 1995 Report at p. 3). Plaintiffs argue that because

16 Klementiev is a "trained and experienced systems analyst, his

17 modeling skills are applicable to various kinds of dynamic,

18 physical processes," including those specifically at issue here

19 involving plutonium production.

20   Defendants argue that while Klementiev might testify

21 regarding his knowledge of computer systems and modeling methods,

22 he should not be able to put himself forward as an expert in the

23 application of those systems to any substantive area. Indeed,

24 the court finds that Klementiev's emphasis is on the modeling and

25 _____

26 [549] Klementiev did not do any epidemiological work for
plaintiffs. Klementiev does not address "health effects." He
27 addresses "source term" which is relevant to "dose."

28 **ORDER RE SUMMARY JUDGMENT-   690**

the mathematical equations in a manner that is detached from the issue of what is likely to have occurred during the plutonium production process at Hanford. Klementiev can crunch the numbers and say what the result is **if** one out of every eight buckets of slugs is transposed, but that is of no value if there is no foundation for such an assumption. Klementiev can tweak HEDR's arithmetic averaging procedure for determining monthly average cooling times, but that is of no value if there is no accounting for what actually occurred during the plutonium production process- i.e. extent of decay within the reactors (reactor bias).

Klementiev's analysis elevates mathematical and numerical form over substance. Klementiev does not have the expertise to substitute for the lack of a substantive evidentiary foundation, nor does he have the expertise in plutonium production processes or radionuclide source term estimation that would make compelling conclusions derived by him from circumstantial evidence. Klementiev's lack of qualification manifests itself in the methodological unsoundness of his source term analysis.

Plaintiffs' emphasis on the contributions of McNeill and Jervis is a concession to Klementiev's lack of expertise in radionuclide source term estimation. Why did Klementiev need McNeill (a nuclear physicist) to oversee his work? Why did Klementiev need to cite the work of Jervis (a radiochemist) when as it turns out, their analyses account for the same alleged errors regarding cooling times and filter efficiency? If McNeill and Jervis somehow supported Klementiev's work, that might be sufficient to salvage it. McNeill and Jervis, however, do not

ORDER RE SUMMARY JUDGMENT-   691

1 support Klementiev's work.

2

3   g.   **Conclusion**

4   The court will grant defendants' motion in limine and

5 exclude Klementiev from testifying about iodine source term

6 estimates.

7

8   4.   **Douglas Stewart**

9   a.   **Introduction**

10   Douglas A. Stewart is a professional meteorologist. He

11 received his Ph.D. in meteorology in 1992 and is employed by

12 Climatological Consulting Corporation, Inc., "providing forensic

13 meteorological services." (Stewart March 1996 Report at pp. 2-

14 3).

15   Stewart is the author of a March 28, 1996 report entitled

16 "Air Dispersion Modeling Issues Related to the Hanford Radiation

17 Litigation." The purposes of this report were: 1) to examine

18 and critique HEDR's air dispersion modeling methodology known as

19 RATCHET (Regional Atmospheric Transport Code for Hanford Emission

20 Tracking); 2) estimate the consequences in terms of I-131

21 concentration and deposition amounts of potential weaknesses in

22 RATCHET; and 3) provide an alternative modeling approach to

23 simulate the transport, dispersion and deposition of I-131 and

24 Pu-239 from which to produce estimates of iodine and plutonium

25 concentration and deposition rates using alternate emissions

26 scenarios and dose estimation techniques. Stewart's alternative

27 model is called RITM (RadioIodine Transport Model).

28
ORDER RE SUMMARY JUDGMENT-   692

1   Stewart prepared a supplemental September 1996 report, "Air

2 Dispersion Modeling Issues Related to the Hanford Radiation

3 Litigation, Supplementary Report." This report, and various

4 other post-March 1996 written revisions prepared by Dr.

5 Stewart[550], were stricken by the court in an order dated

6 November 18, 1996. (Ct. Rec. 858). The report did not meet the

7 supplementation criteria established by this court and instead

8 constituted an effort to revise and improve an existing report

9 (the March 1996 report) in order to shield it from criticism. In

10 striking the supplemental report, this court stated defendants

11 could not use Stewart's **actual** supplemental report or his **actual**

12 post-March 1996 written revisions in cross-examining him, nor

13 could they cite to the supplemental report or to the written

14 revisions. The defendants were not precluded from attacking Dr.

15 Stewart's March 1996 model so long as the source of the attack

16 was not the **documents** stricken by the court's order.

17   In an order dated January 9, 1997, this court denied

18 plaintiffs' motion seeking reconsideration of the order striking

19 Stewart's post-March 1996 work. (Ct. Rec. 884). Consequently,

20 the only report at issue here is Stewart's March 1996 report.

21   At the outset, the court notes that because it is striking

22 Dr. Klementiev's source term analysis, it must also strike Dr.

23

24   [550] June 28, 1996 "Revised Section 6 of Air Dispersion
    Models Related to the Hanford Radiation Litigation;" June 28,
25 1996 "Revised Figure 2 of Air Dispersion Models Related to the
    Hanford Radiation Litigation;" June 28, 1996 "Revised References
26 to Air Dispersion Models Related to the Hanford Radiation
    Litigation;" and June 18, 1996 "Revised Appendix F to Air
27 Dispersion Models Related to the Hanford Radiation Litigation."

28 **ORDER RE SUMMARY JUDGMENT-   693**

1 Stewart's dispersion analysis since it relies upon Klementiev's

2 emission estimates.   (Appendices E and F to Stewart March 1996

3 Report at pp. 76 and 79).[551] That, however, is not the only

4 basis upon which defendants attack Stewart's work.

5

6   **b.   HEDR's RATCHET Model**

7   RATCHET models the movement of I-131 emitted from the

8 Hanford exhaust stacks through the atmosphere to deposition on

9 the ground within the HEDR study area. See generally, Ramsdell,

10 "Regional Atmospheric Transport Code for Hanford Emission

11 Tracking," (January 1994) (PNWD-2224 HEDR).[552] RATCHET is a

12 "Langrangian **puff** model" and can track **individual elements**, such

13 as I-131, that are released from the source. (Stewart Dep. at p.

14 156). A "grid" is superimposed over the HEDR study area and

15 individual iodine releases are followed as they move from grid

16 node to grid node. The movement of the individual releases is

17 modeled on an **hourly** basis. (Id. at p. 108).

18   RATCHET considers hourly variations in wind speed, wind

19 direction, precipitation, terrain, and atmospheric "mixing" of a

20 "puff" as it travels downwind from grid node to grid node.

21 RATCHET is capable of utilizing a large database of time varying

22 and spatially varying input, including winds, spatially varying

23 surface roughness (terrain), and spatially and time varying

24 mixture heights. Each puff is a different age and located

25 _____

26   [551] Stewart used Klementiev's Scenarios 2a and 2b for 1944-
    49. He used Klementiev's Scenarios 5 and 6 for 1950-1960.

27   [552] Hereinafter "Ramsdell 1994." Defendants' Ex. 103.

28 **ORDER RE SUMMARY JUDGMENT-   694**

1 somewhere within the grid where it experiences applicable local

2 meteorological conditions.   (Id. at p. 157).

3   RATCHET must account for the form of iodine emitted from the

4 stacks because the form affects the rate at which iodine is

5 deposited on the ground.   Hanford's iodine emissions took three

6 different forms: gaseous elemental (inorganic); particulate; and

7 gaseous organic.   (Stewart March 1996 Report at pp. 5-6).

8 Gaseous elemental iodine is deposited most quickly on surfaces,

9 while particulate iodine is deposited more slowly.   Gaseous

10 organic iodine tends to remain aloft.   (Stewart Dep. at p. 126).

11 Because these forms of iodine deposit on the ground at differing

12 rates and because airborne iodine continues to decay as it

13 travels through the air, HEDR estimated the extent to which each

14 form comprised Hanford iodine emissions.   (Ramsdell 1994 at p.

15 2.31).

16   RATCHET assumes 27 percent of Hanford emissions was gaseous

17 elemental, 28 percent was particulate, and 45 percent was gaseous

18 organic.   It also assumes these fractions remained constant as

19 the iodine traveled downwind, meaning that slower-depositing

20 iodine transformed to more rapidly-depositing forms as those

21 forms were deposited on the ground.   (Stewart March 1996 Report

22 at p. 6).

23

24   **c.   Stewart's RITM Model**

25   Stewart's model is a Gaussian **plume** model.   It analyzes the

26 movement of iodine along a straight line from its source to

27 deposition.   At his deposition, Stewart agreed with counsel's

28 **ORDER RE SUMMARY JUDGMENT-   695**

1   description of the plume model as modeling along an entire "ray"

2   away from the source, while the "puff" model breaks that into

3   smaller segments and can calculate deposition as the emissions

4   move along the "ray." (Stewart Dep. at pp. 157-58). The "plume"

5   model cannot track from grid node to grid node.

6   The RITM model does not account for diurnal (day and night)

7   variations in atmospheric, or other, conditions because it deals

8   with time variations on a monthly basis. (Id. at pp. 143-44).

9   Because Hanford dissolved fuel (slugs) at night, Stewart

10 developed adjustment factors to correct for his model's

11 assumption of a continuous or average emission of iodine. (Id.

12 at pp. 133-34).

13   The Gaussian plume model requires an assumption of uniform

14 conditions along the entire path of the movement of the iodine

15 emission. This includes uniform wind speed and direction,

16 uniform precipitation, uniform terrain surface roughness, and

17 uniform "mixing depth" within the atmosphere. (Id. at p. 156).

18   In his March 1996 report, Stewart described the effect of

19 his model's inability to accept time-varying emissions and its

20 assumption of uniform conditions:

21   The model produces concentration and deposition
             amounts that are reasonable in view of its
22   simplicity. The inability of the modeling
             framework to accept time-varying emissions
23   has been addressed by developing and applying
             a correction factor to the concentration and
24   deposition patterns that effectively distribute
             the iodine further away from the source. The
25   requirement that the model use constant precipitation
             rates tends to result in an overestimation of
26   deposition fluxes. The spatial distribution
             of the overestimation is difficult to determine
27   with precision, but is estimated to be less than

28 **ORDER RE SUMMARY JUDGMENT-   696**

1   a factor of 2 at distances beyond 30-50Km downwind.
     Closer to the source, the overestimation could be
2   a factor of 4. Thus, the deposition values generated
     by the . . . model should be considered **upper bound**
3   **estimates.**

4   (Stewart March 1996 Report at p. 14)(Emphasis added). Stewart

5   acknowledged the existence of a "wet deposition" bias which

6   caused an "overestimation" of the amount of iodine deposited on

7   the ground.[553]

8   Stewart's model assumes partitioning fractions of 10%

9   elemental (inorganic) iodine, 25% organic iodine, and 65%

10 particulate iodine, leading to "a slightly larger total iodine

11 loading downwind" than through use of HEDR's average fractions.

12 (Id. at p. 6). HEDR assumed that only 28% of the iodine

13 emissions was in particulate form. Thus, under Stewart's

14 assumption and because particulate iodine stays airborne longer,

15 the result is greater deposition further away from the source

16 (the stacks).

17   Stewart's March 1996 model produces **deposition** estimates

18 which exceed the amount of iodine released according to

19 Klementiev's **release** estimates. This applies to all of the years

20 considered (1944-60). (Stewart Dep. at pp. 56-57; 65-66). For

21 example, for 1945, Stewart did not dispute his deposition

22 estimate was 146% of what Klementiev estimated was released for

23 that year. (Id. at p. 57).

24 //

25 //

26 _____

       [553] This is one of the things he tried to correct in his
27 stricken supplemental report.

28 **ORDER RE SUMMARY JUDGMENT-   697**

d. Reliability

(1) Mass Balance Principle

Defendants contend the basic problem with Stewart's analysis is that he has disavowed the iodine deposition estimates produced by his March 1996 model. This is because of the "mass balance" problem described above in which Stewart's March 1996 model shows more iodine **deposited** on the ground than was **released** into the atmosphere. A fundamental principle is that an atmospheric transport model should not deposit more of a substance than is input into the model. (Stewart Dep. at p. 57). Due to the decay of iodine, there cannot be more iodine deposited than was released. (Id. at pp. 55-56).

At his deposition, Stewart testified as follows:

> I think that if you took the numbers for
> . . . my March '96 results, you would
> find that more was deposited out than was
> emitted. That was one of the reasons I
> went back and made revisions that I sub-
> mitted in June [1996] because I couldn't
> stand by the results of the model that
> didn't at least approximate a mass balance.
> There is no rigorous mass balance to the
> approach I've used. And I can explain that
> in more detail if you want. But, certainly
> looking at my March results, I discovered
> something was amiss.

(Id. at pp. 56-57).

The "mass balance" problem was so serious that Stewart testified he could not stand by his March 1996 results. He testified he would have to go to his June 1996 revisions in order to get an endorsement of his work. According to Stewart, he found an "inconsistency" in his model which he corrected because it was the "ethically responsible thing to do." (Id. at pp. 105-

ORDER RE SUMMARY JUDGMENT-   698

1   06).

2   Stewart acknowledged that one "predominant" reason for the

3   "mass balance" problem was the inability of his RITM to account

4   for "time varying emissions" and the consequent need for an

5   adjustment factor. (Id. at pp. 138-39, 256-57). As noted above,

6   the RITM does not account for hourly changes in atmospheric

7   conditions as the iodine moves downwind. Another "predominant"

8   reason for the "mass balance" problem, according to Stewart, was

9   a "coding error" involving a factor which "should have been

10   calculated as a smaller value," therefore causing "slightly

11   larger depositions to be generated." (Id. at pp. 188-89; 256).

12   This coding error was another item which Stewart endeavored to

13   correct in his post-March 1996 revisions which were ultimately

14   incorporated in his stricken September 1996 supplemental report.

15   Essentially, when this court struck his post-March 1996

16   revisions and his September 1996 supplemental report, Dr.

17   Stewart's fate was sealed. Without his post-March 1996

18   revisions, Stewart's March 1996 model cannot survive because of

19   the "mass balance" problem. The only thing before the court is

20   the March 1996 model and it is not scientifically reliable.

21   Stewart claims he had an ethical responsibility to make

22   corrections to his model. While that may be the case, the

23   corrections could not be made at the expense of the court's case

24   management schedule regarding the time for submission of expert

25   reports and the strict conditions placed upon the submission of

26   any supplemental reports. (See Court's January 9, 1997 Order

27   Denying Motion for Reconsideration). This is not an attack upon

28   **ORDER RE SUMMARY JUDGMENT-   699**

1 Stewart's integrity, but the hard, cold fact that deadlines must

2 eventually be met.

3   Plaintiffs make a feeble attempt to defend Stewart's March

4 1996 model, citing deposition testimony which actually reinforces

5 the conclusion that the March 1996 results are scientifically

6 unreliable as a whole:

7   I would feel comfortable in saying that the
           numbers produced by [the March 1996] model

8   with all of its problems are not vastly
           different than the best refinements I got,

9   particularly downstream near Spokane and
           remote areas. **Close to the source, I would**

10   **not rely on those numbers. I would fall**
           short of endorsing that model because I

11   **know it has a mass balance issue which is**
           related to how high the concentrations are

12   **near the source. And if pointed out that**
           the time varying emissions factor used this

13   **plume center line values that were probably**
           inappropriate, I would agree with that.

14
           There are various issues that I feel much

15   **more confident in going beyond the March**
           report, but if the March report is all that's

16   available, I know those numbers are not vastly
           different than the refined numbers again,

17   depending on where you are. **And I would say**
           I feel much more comfortable using the refined

18   **numbers.**

19 (Stewart Dep. at pp. 263-64)(Emphasis added).

20   Recognizing the serious problems with Stewart's March 1996

21 model, plaintiffs attempt to focus attention elsewhere. First,

22 they suggest defendants have violated this court's order

23 prohibiting them from using Stewart's stricken post-March 1996

24 work against him. According to plaintiffs, the source of all

25 defendants' criticisms of Stewart's March 1996 model is Stewart's

26 post-March 1996 revisions. It appears to be true, as plaintiffs

27 point out, that defendants' experts did not offer these

28 **ORDER RE SUMMARY JUDGMENT-**   **700**

1 | criticisms.

2 | In its November 18, 1996 order striking Dr. Stewart's

3 | supplemental report, this court stated:

> Defendants' experts can identify contradiction
> or errors in Dr. Stewart's March 1996 model
> which are derived from their examination of the
> March 1996 model and their own scientific analysis
> or investigation. They simply cannot identify
> Dr. Stewart's post-March 1996 documents as the source
> of any contradiction or error in the March 1996 model.
> If Dr. Stewart cannot rely on his supplemental report
> and his written revisions, his admissions concerning
> error and the need for analytical refinement cannot
> be used against him.

10 | (Ct. Rec. 858 at pp. 9-10).

11 | The court's concern was with defendants and/or their experts

12 | using Stewart's stricken **documents**. The court stated defendants

13 | could not use the **actual** supplemental report and the **actual** post-

14 | March 1996 written revisions in any cross-examination of Dr.

15 | Stewart. Defendants were also prohibited from citing to

16 | Stewart's supplemental report or to his written revisions. (<u>Id</u>.

17 | at p. 9).

18 | There is no indication defendants confronted Stewart at his

19 | deposition with his actual supplemental report and his actual

20 | post-March 1996 written revisions. Defendants' briefs with

21 | regard to this motion in limine do not cite any of those

22 | materials. Realistically, of course, that does not mean

23 | defendants' counsel came up on their own with the criticisms they

24 | have leveled against Stewart's analysis. By the time of

25 | Stewart's deposition, defendants' counsel had in their possession

26 | his supplemental report and his post-March 1996 revisions. It is

27 | too far-fetched to say counsel did not use those materials in

28 | **ORDER RE SUMMARY JUDGMENT-   701**

1 formulating questions for the deposition and formulating

2 arguments to be included in the motion in limine.

3   However, there is nothing wrong with that. To Stewart's

4 credit, he was entirely forthcoming about the deficiencies in his

5 March 1996 model, deficiencies which he pointed out and

6 endeavored to correct in his post-March 1996 work. The

7 defendants did not need to wave the supplemental report or the

8 post-March 1996 revisions in Stewart's face. Form should not be

9 elevated over substance here. The indisputable fact is there are

10 deficiencies in Stewart's March 1996 model. Those deficiencies

11 should not be swept under the rug. They are legitimate points of

12 discussion in the analysis of whether Stewart's March 1996 model

13 is scientifically reliable.

14   The court's concern was that the post-March 1996 revisions

15 not be considered an automatic concession on Stewart's part or

16 plaintiffs' part that the March 1996 model, **by itself**, could not

17 survive <u>Daubert</u> scrutiny. The plaintiffs and Stewart were

18 entitled an opportunity to show that the March 1996 model,

19 despite any deficiencies and despite the subsequent revisions,

20 was scientifically reliable. Had they been able to do that, then

21 the defendants would not have been able to refer at trial to

22 Stewart's post-March 1996 work in an effort to impeach him.

23   Plaintiffs contend the deficiencies in Stewart's March 1996

24 RITM model may affect the **weight** a jury should afford the model,

25 but do not affect its admissibility. This argument ignores the

26 fact Stewart is not willing to stand by his March 1996 results.

27 If Stewart is unwilling to stand by those results, he obviously

28 **ORDER RE SUMMARY JUDGMENT-**   **702**

1   does not have "good grounds" for those results. His March 1996

2   analysis is so flawed, it must be excluded.

3

4   **(2) Wet Deposition Bias**

5   While the "mass balance" problem is the most egregious flaw

6   in Stewart's March 1996 model and by itself requires exclusion of

7   the model, defendants contend there are other flaws which also

8   require exclusion. In his March 1996 report, Stewart

9   acknowledged a "wet deposition bias" in his model. At his

10   deposition, he explained this was the result of his model's use

11   of a "time-constant" precipitation rate:

12   Say it rains for three days or a week
               during the month and it rains at the rate

13   of an inch a day. Then you have seven inches
               in a 28 day month, say. RITM would have

14   to be provided with a rainfall estimate of
               seven inches over 28 days which is a

15   different rainfall rate. **And that rainfall**
               rate would end up depositing <u>more</u> particulate

16   **iodine than if we applied . . . one inch per**
               day over a week and then applied no wet

17   **deposition over three weeks.**

18   (<u>Id</u>. at pp. 148-49)(Emphasis added).

19   Stewart indicated the bias tends to result in larger

20   deposition values, particularly near the source (the stacks).

21   Furthermore, he stated the bias cannot be precisely quantified

22   without "a set of long-term time-dependent simulations using a

23   RATCHET-like model." (Stewart March 1996 Report at p. 9). This

24   is because RATCHET, as noted above, does not employ a constant

25   precipitation rate.

26   The "wet deposition" bias is something which Dr. Stewart

27   endeavored to correct in his stricken post-March 1996 work.

28   **ORDER RE SUMMARY JUDGMENT-**   **703**

1 Stewart testified he would not stand by the precipitation bias

2 estimates contained in his March 1996 report because he did "more

3 refined bias estimates later . . . ." (Stewart Dep. at p. 150).

4 The "wet deposition bias" is yet another example of

5 methodological unsoundness, further warranting exclusion of

6 Stewart's March 1996 results. The "wet deposition" bias

7 contributes to the "mass balance" problem wherein more iodine is

8 deposited than released.

9

10   **(3) Partitioning of Iodine/Transformation**

11   The precipitation bias is especially significant because of

12 the assumption in Stewart's March 1996 model that 10% of the

13 iodine was gaseous elemental (inorganic), 25% was gaseous

14 organic, and 65% was particulate. According to Stewart,

15 particulate iodine deposits more rapidly by wet processes.

16 (Stewart Dep. at p. 123). Stewart testified his assumptions

17 regarding iodine fractionation are derived from work done by Dr.

18 Robert Jervis, another of the plaintiffs' experts. Stewart

19 testified he received those figures from Jervis in a telephone

20 conversation. (Id. at p. 163).

21   Defendants cite Jervis' November 1995 report, "Evaluation of

22 Radiochemical Aspects of HEDR," wherein he opined that "the

23 composition of released I-131 before 1950, when sand bed filters

24 and silver reactors were deployed to reduce particulate and

25 inorganic iodine, could have been: 20% particulate, 70%

26

27
28 **ORDER RE SUMMARY JUDGMENT-   704**

1 inorganic, 10% organic." (Jervis 1995 Report at p. 6).[554]

2 Defendants contend Stewart's assumption is inconsistent with the

3 figures in Jervis' report and there is nothing to document a

4 telephone conversation between Stewart and Jervis during which

5 Jervis endorsed the figures used by Stewart.

6   Plaintiffs assume a conversation did in fact occur between

7 Jervis and Stewart, although there is no affidavit from Jervis

8 confirming such. Plaintiffs contend defendants misrepresent the

9 figures in Jervis' 1995 report because those figures pertain only

10 to the composition of the iodine **when it leaves the stack, and**

11 **not to its changing composition after it leaves the stack.**

12 Indeed, Jervis stated in his report:

13   . . . the composition distribution among radioiodide
            species in a dispersing plume moving away from the
14   stacks would have been continuously changing with
            distance because of the very different behaviour
15   of species during atmospheric transport, and, dry and
            wet deposition over long distances (tens of miles) of
16   transport.

17   . . . another effect to be considered is the
            probability that some elemental gaseous iodine, being
18   inherently so chemically reactive, would readily
            adsorb onto ambient, submicron ambient atmospheric
19   aerosol particles downwind from the stacks and be
            transported and deposited in this state. Ambient
20   aerosol, mainly tiny soil grains, is ubiquitous even
            in remote atmospheres.

21
   (Jervis 1995 Report at pp. 6-7).
22
        Jervis appears to suggest that because of this, the
23
   percentage of iodine ultimately deposited in particulate form
24
   _____

25   [554] Jervis stated that after 1950 when the absorber beds
   were installed and a stacked filter column added, most
26 particulate iodine would have been eliminated, resulting in an
   approximate composition of 0-5% particulate, 50-60% inorganic,
27 and 30-40% organic. (Jervis 1995 Report at p. 6).

28
   ORDER RE SUMMARY JUDGMENT-   705

1 might be more than the percentage found at the stack.

2 Nevertheless, the court remains suspicious why plaintiffs made no

3 effort to have Jervis confirm in writing the specific figures

4 used by Stewart and offer some explanation for those figures.

5   Plaintiffs counsel attack the partitioning assumption used

6 by RATCHET (27% gaseous elemental (inorganic), 28% percent

7 particulate, 45% gaseous organic). Citing HEDR documents, they

8 say that prior to 1950 the iodine released was primarily in

9 elemental form, with a very small portion in organic form.

10 According to plaintiffs, RATCHET's partitioning assumption is

11 based on studies conducted in 1964, after the installation of

12 silver reactors, which showed a decrease in the amount of

13 elemental (inorganic) iodine and an increase in the amount of

14 organic iodine. Plaintiffs assert it is unreasonable for RATCHET

15 to have applied a partitioning assumption including such a high

16 percentage of organic iodine (45% organic) to releases prior to

17 1950 and before the advent of the silver reactors. Because

18 organic iodine stays aloft longer, it is more likely to have been

19 blown completely out of the HEDR study area. Consequently, the

20 result is less total iodine deposited in the study area and

21 decreased doses.

22 **Plaintiffs' argument regarding organic iodine is not**

23 **supported by any specific reference to work done either by**

24 **Stewart or Jervis.** As noted above, however, Jervis opines that

25 prior to the installation of sand bed filters and silver

26 reactors, the composition of released I-131 (prior to any

27 chemical transformation in the atmosphere) could have been 20%

28 **ORDER RE SUMMARY JUDGMENT-   706**

1   particulate, 70% inorganic (elemental) and 10% organic. Jervis

2   seemingly opines that following release, the amount of

3   particulate could have increased because of the adsorption[555] of

4   elemental (inorganic) iodine onto submicron ambient particles.

5   However, he does not say anything about a change in the organic

6   iodine composition due to chemical transformation in the

7   atmosphere. The 10% figure for organic iodine may constitute

8   some support for an attack upon RATCHET's assumption of 45%

9   organic iodine.

10   For the period after 1950 and installation of the sand bed

11 filters and silver reactors, Jervis says the composition of the

12 iodine released at the stack would more likely be 0 to 5%

13 particulate, 50-60% elemental (inorganic) and 30 to 40% organic.

14 Clearly, the organic figure here (40%) is more in line with

15 RATCHET's assumption of 45% organic iodine. However, according

16 to Stewart, Jervis apparently was willing to later modify his

17 figures to 25% organic, 65% particulate, and 10% inorganic

18 elemental for releases both before and after 1950.

19   The court must determine whether Stewart has a scientific

20 basis for **his** partitioning assumption. Unless the court simply

21 accepts that Jervis provided Stewart with this partitioning

22 assumption and that there are good grounds supporting it, all the

23 court is left with is drawing inferences from the figures and

24 comments provided in Jervis' 1995 report. Jervis' report

25 _____

26   [555] Adhesion of an extremely thin layer of molecules (as of
    gases, solutes or liquids) to the surface of solid bodies or
27 liquids with which they are in contact.
28
    ORDER RE SUMMARY JUDGMENT-   707

1   arguably provides some support for an increase in the particulate

2   fraction, but the question is just exactly how much? Stewart

3   says Jervis increased the particulate fraction by 45% for the

4   period prior to 1950 (20% to 65%) and by 60 to 65% for the period

5   after 1950 (0 to 5% increased to 65%). These are significant

6   increases.

7   The 25% organic iodine figure Stewart says Jervis provided

8   him is the halfway point between the 10% and 40% figures Jervis

9   provided in his report regarding the pre-1950 and post-1960

10   periods. Perhaps this is a reasonable inference for how the 25%

11   figure was derived. Once again, there is nothing from Jervis

12   confirming his approval of a 25% figure.

13   Ultimately, what needs to be kept in mind is the bottom

14   line. Stewart's partitioning assumption (10% elemental

15   inorganic; 65% particulate; and 25% organic), as plugged into his

16   March 1996 model, produced results in violation of the "mass

17   balance" principle: more iodine deposited on the ground in the

18   HEDR study domain than released from the stacks. While there may

19   be valid reasons to challenge RATCHET's partitioning assumptions

20   (27% elemental inorganic; 28% particulate; and 45% organic) and

21   its results- 56% of the total iodine released deposited in the

22   HEDR study domain, 10% decayed in the study area, and 34% left

23   the study area[556]- the fact is those results are not a violation

24   of the "mass balance" principle. The amount of iodine released

25   _____

26   [556]   These are the mean results reported in J.V. Ramsdell,
     Jr., et al., "Atmospheric Dispersion and Deposition of I-131
     Released from the Hanford Site," 71 **Health Physics** 568 (1996),

27   p. 575.

28   **ORDER RE SUMMARY JUDGMENT-   708**

1 does not exceed the amount deposited.

2   In his March 1996 report, one of the "potential

3 shortcomings" Stewart identified regarding RATCHET was its

4 assumption that "the fraction of radioiodine existing in [the]

5 three forms is constant with time." (Stewart Report at p. 4). At

6 his deposition, Stewart testified that it was a shortcoming "not

7 . . . easily resolved by any effort, either HEDR's effort or my

8 effort." (Stewart Dep. at p. 108). Indeed, Stewart ultimately

9 decided to assume for his RITM model that the fraction was

10 constant with time:

11   In summary, the RATCHET application to the
          HEDR project produces conservative estimates
12   of radioiodine concentration and deposition
          flux distributions **with respect to the constant**
13   **treatment of radioiodine partitioning into its**
          components. This is probably a justifiable
14   treatment in view of the uncertainty in actual
          radiochemical transformations, and has been
15   adopted for my calculations as well. Whether
          or not a more realistic radiochemical transformation
16   scheme could have been adopted is beyond my
          expertise. The proportions of radioiodine in the
17   gas and particulate phases chosen for the RATCHET
          study may underestimate the abundance of particulate
18   iodine, according to [Jervis] (1995). For this
          reason, the regional concentration and deposition
19   fluxes presented in Appendix F use a split that
          is adjusted to reflect greater particulate emissions.

20 (Stewart Report at p. 14)(Emphasis in text). For Appendix F,

21 Stewart used what he says is Jervis' "split"- 10% gaseous

22 elemental iodine, 25% gaseous organic, and 65% particulate-

23 constant over time.

24   Defendants contend Stewart's decision was motivated by his

25 realization that HEDR's assumption of a constant partitioning

26 actually biases HEDR's deposition numbers upward. In his report,

27
28 **ORDER RE SUMMARY JUDGMENT-   709**

1 Stewart stated:

2   Maintaining a constant partitioning of radioiodine
            implicitly assumes a transformation between components.
3   This transformation converts the more stable forms of
            radioiodine that might otherwise travel considerable
4   distances downwind [organic gaseous iodine and
            particulate iodine], to the more reactive form
5   [elemental gaseous iodine] that is readily deposited.
            The spatial scales of the modeling domain coupled with
6   the residence times of the three radioiodine components
            (which depend on the climatological frequency of
7   precipitation) produce a **net excess** of concentration
            and deposition flux within the modeling domain [aka
8   HEDR Study Area]. Certainly, these excesses become
            deficits further downwind, but the distance scale at
9   which this occurs is larger [than] the Hanford modeling
            domain.

10 (Stewart March 1996 Report at p. 6)(Emphasis added).[557]

11   Defendants apparently suggest Stewart's decision to stick

12 with a constant partitioning reflects nothing more than a desire

13 to insure the highest deposition numbers. Defendants assert

14 Stewart could have "easily accounted" for the **alleged** shortcoming

15 of the constant partition rate, but opted not to once he realized

16 the constant partition rate actually produced higher numbers.

17   At his deposition, Stewart testified his model (RITM) was

18 configured to incorporate a "more explicit transformation

19 scheme," but he opted not to do so because of the effort

20 involved. (Stewart Dep. at p. 128). Later, at his deposition,

21 Stewart conceded use of the constant partition rate was not

22 really a shortcoming because no one, including HEDR, knew how to

23 incorporate the transformations into the model, whether it be

24

25 _____

   [557] At his deposition, Stewart reiterated that in assuming
26 the fraction remains the same, HEDR implicitly allows the less
   reactive forms or less rapidly deposited forms to be converted
27 into a more rapidly deposited form. (Stewart Dep. at p. 126).
28
   ORDER RE SUMMARY JUDGMENT-   710

1 RATCHET or RITM. (Id. at p. 142).[558]

2   The fact that in his report Stewart identified the constant

3 partition assumption as a "potential shortcoming" is not

4 necessarily an indication that he was simply out to produce

5 higher numbers for the plaintiffs. The fact is Stewart ended up

6 using a constant partition rate which defendants do not claim is

7 scientifically unreliable in itself, although they take issue

8 with the numbers Stewart used (10% gaseous elemental iodine, 25%

9 gaseous organic, and 65% particulate).

10

11   **(4) Validation**

12   Defendants assert an additional indication of the

13 unreliability of Stewart's model is his failure to test his model

14 results against any measured environmental data (vegetation, milk

15 concentration, etc.). Stewart testified he instead compared his

16 _____

17   [558] Stewart was a bit hazy about the impact of **not using a
constant partition rate**. Stewart testified that from the
sensitivity studies he performed, "it appeared as though if the

18 individual components were allowed to vary that you would end up
with **lower**, slightly lower concentrations and deposition amounts

19 in the [HEDR study] area." (Stewart Dep. at p. 127)(Emphasis
added).

20   As noted, Stewart testified that **using the constant
partition factor** would produce **higher** amounts within the study

21 area, but these excesses would become deficits outside of the
study area. Stewart seemingly testified that this would be the

22 case with "less precipitation." However, if there was "more
precipitation," he testified it would lead to a deficit in

23 deposition amounts closer to the source. (Stewart Dep. at p.
131). It is not clear what the concern is if the deficit occurs

24 **outside** of the study area.
      It appears this is a case of Stewart speculating that

25 something is wrong and not knowing how to fix it. Indeed,
Stewart acknowledged he did not have enough expertise to justify

26 a more explicit transformation scheme and that he did not "know
enough about that complicated process to pursue it." (Id. at p.

27 132).

28
   ORDER RE SUMMARY JUDGMENT–   711

1 results with RATCHET's results. According to Stewart, he assumed

2 the validity of his model by "ballpark comparisons" with RATCHET,

3 and if his results had been "grossly in error," he would have

4 decided whether his approach was valid. (Stewart Dep. at pp. 80-

5 86). Stewart testified that since his model reproduced HEDR's

6 results "with some degree of confidence," he concluded his

7 approach was adequate and justifiable. (Id. at p. 87).

8   Defendants contend Stewart's validation of his RITM model by

9 comparison to the RATCHET model results does not make sense since

10 RITM is offered as an alternative to RATCHET. Defendants contend

11 Stewart's failure to validate his model results with measured

12 environmental data is unscientific and requires exclusion of

13 those results.

14   The subject of an expert's testimony must be scientific

15 **knowledge.** In order to qualify as "scientific knowledge," an

16 inference or assertion must be derived by the scientific method

17 and proposed testimony must be supported by appropriate

18 **validation-** i.e. good grounds, **based on what is known.** Daubert

19 I, 509 U.S. at 590. The requirement that an expert's testimony

20 pertain to "scientific knowledge" establishes a standard of

21 evidentiary reliability or "trustworthiness." Id. and n. 9.

22   Rather than defending what Stewart did or did not do in the

23 way of validation, the plaintiffs devote a portion of their

24 response brief to arguing that RATCHET's results have fared

25 poorly in validation exercises. Assuming RATCHET indeed fared so

26 poorly, one has to ask what that says about Stewart's RITM

27 results since he assessed the validity of his results on how well

28 **ORDER RE SUMMARY JUDGMENT-   712**

they compared with RATCHET's results. Furthermore, if RATCHET

fared so poorly, that seems all the more reason for Stewart to

have undertaken his own independent validation of the RITM

results (aka model predictions) using the measured environmental

data.[559]

Plaintiffs' attack on RATCHET does nothing to redeem

Stewart's March 1996 RITM results.

(5) **Daubert** Criteria

Plaintiffs claim that because the work Stewart has performed

─────────────────

[559] The plaintiffs retained Jesse L. The', Ph.D., a
professional engineer, to compare vegetation concentration
measurements against concentrations predicted by using the
deposition fluxes generated by Stewart's RITM. The report/
affidavit of The', "Hanford Litigation-Radioiodine Vegetation
Concentration Comparison," (Foulds Ex. 137), was prepared in July
1997, after Stewart had completed his post-March 1996 work,
including his September 1996 supplemental report. However, the
report/affidavit indicates The' used only the deposition fluxes
from Stewart's March 1996 report. Perhaps The' confined himself
to the March 1996 results considering that in November 1996 this
court had already stricken Stewart's supplemental work.
Plaintiffs conclusorily contend the work of The' finds
Stewart's model accurately predicts historically measured iodine
ground deposition. However, the plaintiffs do not elaborate on
this in their response brief. In their reply brief, defendants
completely ignore the work of The'. Perhaps the reason is that
his work was submitted so long after Stewart's March 1996
results. It was actually submitted as part of plaintiffs'
response brief to the motion in limine. The report/affidavit of
The' indicates he did not start his review activities until **June
1997**. Therefore, it appears defendants had no opportunity to
depose The'.
Furthermore, a review of the report/affidavit indicates
comparison of Stewart's RITM predictions with vegetation
concentration measurements was incomplete and more work needed to
be done in several different respects. The report/affidavit from
The' is a concession by plaintiffs of the need for validation of
Stewart's results. Whatever value this belated validation effort
may have, it is not enough to overcome Stewart's disavowal of his
March 1996 results because of violation of the "mass balance"
principle.

ORDER RE SUMMARY JUDGMENT-   **713**

1 "falls squarely within the areas of study he has emphasized

2 throughout his career," it "grows 'naturally and directly' out of

3 research conducted independent of this litigation." This ignores

4 the fact that the specific model developed by Stewart- the RITM-

5 was clearly developed for the purpose of this litigation.

6 According to Stewart: "A Gaussian modeling approach has been

7 **developed** and applied to generate concentration and deposition

8 fluxes to enable examination of **alternative emissions and dose**

9 **estimation procedures.**" (Stewart Report at p. 14)(Emphasis

10 added). Stewart was retained by counsel to provide this

11 alternative modeling approach. (<u>Id</u>. at p. 1). RITM did not

12 exist prior to this litigation.

13   Plaintiffs contend the "general principles behind Dr.

14 Stewart's work, specifically the efficacy of the Gaussian

15 methodology have been thoroughly scrutinized by the scientific

16 community." They claim RITM uses methodologies "similar" to

17 those in previously published and peer-reviewed models and that

18 "some" parameters in RITM are identical to those in previous

19 models used by Stewart. Nonetheless, Stewart acknowledged the

20 RITM model **itself** has not been peer-reviewed. (Stewart Dep. at

21 pp. 75, 230 and 258).

22   Independent research and peer review are the two principal

23 ways for showing that evidence satisfies the scientific

24 reliability prong of <u>Daubert</u>. These criteria do not weigh in Dr.

25 Stewart's favor. Because RITM was specifically developed for

26 this litigation and has not been peer reviewed, it is no surprise

27 that it has not received anything which can be termed "general

28 **ORDER RE SUMMARY JUDGMENT-   714**

1 acceptance" within the scientific community.

2   Plaintiffs argue Stewart's model can be easily tested

3 against defendants' vegetation data. Even if that is the case,

4 testing cannot erase the "mass balance" problem arising from the

5 March 1996 model.

6

7   **e. Fit/Relevancy**

8   The best Stewart can say about his March 1996 results is

9 they are not "wildly" unreliable (Stewart Dep. at p. 184) "upper

10 bound" estimates. (Stewart March 1996 Report at p. 14). Such

11 estimates are of no assistance to a trier of fact in determining

12 realistically how much iodine was deposited on the ground and in

13 turn, the dose likely received by a particular plaintiff.

14 Because these estimates are "unreliable," they do not "fit" the

15 pertinent inquiry.

16

17   **f. Conclusion**

18   Dr. Stewart will be excluded from testifying at trial.

19 First, Stewart's analysis is based on the source term estimates

20 of Dr. Klementiev which are not scientifically reliable.

21 Exclusion of Klementiev's work requires exclusion of Stewart's

22 work. Secondly, Stewart's March 1996 atmosphere transport model

23 is not scientifically reliable and produces results which are of

24 no assistance to a jury.

25   Exclusion of Stewart's RITM model does **not** necessarily mean

26 RATCHET is scientifically reliable or otherwise not subject to

27 legitimate criticism. However, the alleged shortcomings in

28 **ORDER RE SUMMARY JUDGMENT-   715**

RATCHET identified by plaintiffs and Dr. Stewart do **not** make

Stewart's RITM model any more reliable. These alleged

shortcomings are not enough to save Stewart's work. If RATCHET

is found to be unreliable, then its results will also be of no

assistance to a jury.

5. Douglas Crawford-Brown

a. Introduction

Douglas Crawford-Brown is a Professor of Environmental

Physics in the Department of Environmental Sciences and

Engineering at the University of North Carolina at Chapel Hill.

He is also the Director of Environmental Studies there.

Crawford-Brown holds a Ph.D. in health physics and nuclear

science from the Department of Nuclear Engineering at the Georgia

Institute of Technology.

In November 1995, Crawford-Brown prepared a report entitled

"Radiation Doses Received by the Population Surrounding the

Hanford Reservation from Releases of Radioiodine into the

Atmosphere in the Period 1944 to 1960." In an affidavit included

with his report, Crawford Brown stated:

> The following report contains the results of
> my calculations of radiation doses from radioiodine
> (I-131) received by the population in areas surrounding
> the Hanford Reservation during the period 1944 to
> 1960. **In all cases, the airborne concentrations and
> rates of depositions onto surfaces were not calculated
> by me, but provided by Dr. Douglas Stewart of
> Climatological Consulting Corporation. The doses
> calculated in this report, therefore, are conditional
> upon the airborne concentrations and rates of
> deposition described at the beginning. I have made no
> independent review of those environmental character-
> izations.**

ORDER RE SUMMARY JUDGMENT-   716

(Crawford-Brown November 1995 Affidavit at p. 2)(Emphasis added).

The airborne concentrations and rates of deposition to which Crawford-Brown refers are those found in Stewart's **November 1995 report.** (Stewart Dep. at pp. 12-14). Stewart's **March 1996 report** bore the same title as his November 1995 report- "Air Dispersion Modeling Issues Related to the Hanford Litigation." Obviously, Crawford-Brown submitted his November 1995 report prior to Stewart's submission of his March 1996 report. However, the results from Stewart's November 1995 report are essentially the same as those in his March 1996 report, the difference being the November 1995 results were reported on an annual basis whereas the March 1996 results were reported on a monthly basis. (Id. at pp. 242-43).

b. Crawford-Brown's Methodology

Crawford-Brown's dose calculations, as found in his report, "are based on the airborne concentrations and deposition rates designated Scenario 6 by Dr. Stewart." (Crawford-Brown November 1995 Report at p. 5). This is a reference to Stewart's "Scenario **6b**" results which are based on Klementiev's Scenario 2b emission (source term) estimates for 1944-49 and his Scenario 6 emission (source term) estimates for 1950-60.

Crawford-Brown developed calculations to convert Stewart's estimates of iodine concentrations and deposition into **thyroid** doses. Crawford-Brown considered the following pathways which he referred to as the "major exposure pathways:" inhalation, ingestion of fruits, ingestion of vegetables, and ingestion of

ORDER RE SUMMARY JUDGMENT-   717

1 dairy products (specifically milk and eggs).   (Crawford-Brown

2 November 1995 Report at p. 5).

3   Crawford-Brown derives his thyroid **inhalation** dose by

4 multiplying an **inhalation** dose factor by Stewart's estimates of

5 iodine concentrations in the air.[560]   This inhalation dose

6 factor varies depending on age.   Crawford-Brown has six different

7 age categories (0-0.5 years; 0.5-2 years; 2-7 years; 7-12 years;

8 12-17 years; adult ages).   (Crawford-Brown November 1995 Report

9 at pp. 5-7).   Crawford-Brown states the methodology used by him

10 for calculating thyroid inhalation dose "is essentially the same

11 as that employed in the HEDR assessment . . . since this is the

12 standard methodology used in the radiation protection practice."

13 The exception is that Crawford-Brown removes the "resuspension"

14 factor since he claims "this does not contribute strongly to dose

15 and simplifies calculations."   (Crawford-Brown November 1995

16 Report at p. 5).   "Resuspension" refers to the movement of

17 material back into the atmosphere once it is settled on the

18 ground.   (Crawford-Brown Dep. at p. 46).

19   Crawford-Brown calculates a thyroid **ingestion** dose for **fruit**

20 **and leafy vegetables** by multiplying an ingestion dose factor by

21 Stewart's estimates of the amount of I-131 deposited on the

22 ground.   This ingestion dose factor varies among age groups.

23 Crawford-Brown provides an **ingestion** dose factor for the same six

24 _____

25   [560]   Stewart evaluated both "ground level airborne
   concentrations" **and** total deposition amounts (actually deposited
26 on the ground).   (Stewart March 1996 Report at p. 79).   Stewart's
   "mass balance" problem is something which affects airborne
27 concentration estimates and deposition estimates alike.

28 **ORDER RE SUMMARY JUDGMENT-   718**

1 age groups for which he provides an **inhalation** dose factor. (<u>Id</u>.

2 at pp. 8-11).

3   From **<u>his</u> fruit and leafy vegetable doses**, Crawford-Brown

4 calculates a **total dose** which includes fruits, leafy vegetables

5 **and milk and eggs.**[561] In other words, from his fruit and leafy

6 vegetable doses, he extrapolates to a total dose. He does this

7 by deriving a "fruit-to-total dose ratio" or "conversion factor"

8 in which he adds the **HEDR** "fruit and leafy vegetables" percentage

9 contribution to dose, to the **HEDR** "milk and eggs" percentage

10 contribution to dose. He then divides by the percentage

11 represented by the "fruit and leafy vegetables" contribution.

12   The percentage contributions calculated by **HEDR** are found in

13 Farris, et al., "Atmospheric Pathway Dosimetry Report, 1944-

14 1992," (October 1994)(PNWD-2228 HEDR), Table 4.4 at pp. 4.44 and

15 4.45.   Table 4.4 lists the "Percent Contribution to Thyroid Dose

16 by Exposure Pathway" for **1945** for **males** residing in Richland and

17 Eltopia.   The table is broken down into age groups: less than 1

18 year old; 1-4 years old; 5-9 years old; 10-14 years old; 15-19

19 years old; and 20-34 years old.   Three different feeding regimes

20 are considered: "Milk Regime 1" pertains to individuals who

21 received their milk from a backyard cow which fed on pasture

22 grass; "Milk Regime 4" pertains to individuals who received their

23 milk from a backyard cow which fed on stored feed; "Commercial

24 Food" pertains to individuals who drank processed milk bought

---

[561] Crawford-Brown stated that for the "milk and eggs"
category of consumption, he relied "entirely" on the methodology
employed by HEDR. (Crawford-Brown November 1995 Report at p.
11).

ORDER RE SUMMARY JUDGMENT-   719

1 from a grocery store. For each feeding regime, ten different

2 exposure pathways are considered: external, inhalation, beef,

3 leafy vegetables, other vegetables, fruit, grain, poultry, eggs

4 and milk. Milk constitutes the majority of the dose in most

5 cases.[562]

6   For a Richland male less than one year old who got his milk

7 from cows grazing on fresh pasture, the combined fruit and leafy

8 vegetables contribution is 12.5% (12.4% + 0.1%), while the

9 combined milk and eggs contribution is 84.4% (82.8% + 1.6%).

10 Under Crawford-Brown's formula, these two figures are added

11 together (12.5% + 84.4% = 96.9%), the sum of which is then

12 divided by the fruit and leafy vegetables percentage of 12.5%

13 (96.9%/12.5%) to arrive at a "fruit-to-total dose ratio" or

14 "conversion factor" of 7.8.[563]

15   7.8 is the "conversion factor" Crawford-Brown arrived at for

16 **all** individuals, males or female, ages one and less, residing in

17 the HEDR study domain, who received their milk from cows grazing

18 on fresh pasture ("Regime 1").[564] Using the HEDR percentage

19 _____

20   [562] Crawford-Brown considers only the inhalation dose
pathway and the fruit, leafy vegetables, milk, and egg dose
pathways. He does not consider the external pathway or the other
21 ingestion dose pathways, which HEDR agrees are not as
significant. Defendants do not mount a <u>Daubert</u> attack on
22 Crawford-Brown's inhalation dose factor.

23   [563] According to Crawford-Brown, the result is a conversion
factor from annual dose for leafy vegetables and fruits **only** to
24 an annual dose from all food sources, which is dependent on age
and regime. (Crawford-Brown November 1995 Report at p. 12).

25   [564] This conversion factor is derived not only from a
26 Richland male age one year or less who got his milk from a cow
grazing on fresh pasture, but also from an **Eltopia** male age one
27 year or less who got his milk from a cow grazing on fresh

28 **ORDER RE SUMMARY JUDGMENT-**   **720**

figures, Crawford-Brown also provides conversion factors for a 1-4 age group, 5-9 age group, 10-14 age group, 15-19 age group, and 19 and over age group, all who received their milk from cows grazing on fresh pasture ("Regime 1").

Using the HEDR percentage figures, Crawford-Brown likewise provides conversion factors for each of these age groups where milk was received from cows fed stored feed ("Regime 4"). (Crawford-Brown November 1995 Report at p. 12). Once again, these conversion factors are intended to apply to **all** individuals within the HEDR study domain and for additional years of exposure beyond 1945, even though they are derived from figures which pertain only to the dose received in 1945 by males residing in either Richland or Eltopia.

The "fruit and leafy vegetable" doses which Crawford-Brown derives from **his ingestion dose factor**[565] are then multiplied by these "conversion factors" or "fruit-to-total dose ratios" to arrive at a **total ingestion dose**. Defendants provide the following example: if Crawford-Brown estimated a "fruit and leafy vegetables" dose of 10 rads for a Richland infant less than

---

pasture. (See Farris 1994 at Table 4.4, pp. 4.44-4.45). The Eltopia figures are slightly different than the Richland figures: fruit and leafy vegetables combination of 11.9% (11.8% + 0.1%) and milk and eggs contribution of 85.6% (84.1% + 1.5%). The Eltopia figures **alone** produce a conversion factor of 8.2 (97.5%[85.6% + 11.9%]/11.9%). Crawford-Brown takes a "weighted average of this conversion factor for the Richland and Eltopia areas, with equal weights," asserting that because the values for the two areas are similar, the choice of weighting factor is not a significant source of uncertainty. (Crawford-Brown November 1995 Report at p. 12).

[565] Crawford-Brown refers to this as his "Equation 4" found at p. 11 of his November 1995 Report.

ORDER RE SUMMARY JUDGMENT-   721

1 year old, he would multiply the 10 rads by his 7.8 "conversion factor" to arrive at a **total ingestion dose** of 78 rads (10 x 7.8).

Crawford-Brown's **inhalation** and **ingestion** doses are added together to arrive at the total thyroid dose for an individual at a particular location. In his report, Crawford-Brown offers **average** thyroid doses received in 1945: 1) by a 1-year old who drank milk from a backyard cow fed pasture grass or stored feed; 2) by a 5-year old who drank milk from a backyard cow fed pasture grass or stored feed; and 3) by an adult who drank milk from a backyard cow fed pasture grass or stored feed. In his report, Crawford-Brown explains how his work could be used "to isolate doses to individuals at specific locations" and how doses could be calculated for years other than 1945. (Crawford-Brown November 1995 Report at p. 13).

One of the exhibits used at Crawford-Brown's deposition was some notes he prepared showing what his thyroid **ingestion** doses would be versus HEDR's ingestion doses, using the **same** estimate of I-131 deposited on the ground. This estimate comes from HEDR, specifically Farris 1994 at p. B.8, Figure B.1. (Defendants' Ex. 22).

For an adult residing in Eltopia and drinking milk from a cow grazed on fresh pasture, Crawford-Brown's ingestion dose calculation was 89 rads, while HEDR's was 6 rads (a 1480% increase). For an adult residing in Eltopia and drinking milk from a cow fed stored feed, Crawford-Brown's dose calculation was 15 rads, while HEDR's was 4 rads (a 380% increase). For a five

ORDER RE SUMMARY JUDGMENT-   722

year old residing in Eltopia and drinking milk from a cow grazed
on fresh pasture, Crawford-Brown's ingestion dose calculation was
256 rads while HEDR's was 25 rads (a 1020% increase). For a five
year old residing in Eltopia and drinking milk from a cow fed
stored feed, Crawford-Brown's ingestion dose calculation was 48
rads while HEDR's was 9 rads (a 533% increase). Defendants note
that Crawford-Brown's doses are 4 to 15 times higher than HEDR's
doses.[566]

c. Reliability

Defendants contend Crawford-Brown's dose calculations should
be excluded for two reasons: 1) he relies on Stewart's iodine
transport numbers as the basis for his dose estimates; and 2) his
"fruit-to-total dose ratios" or "conversion factors" are based on
erroneous assumptions and mathematical errors.

(1) Use of Stewart's Concentration and Deposition Estimates

The court is excluding Stewart's concentration and

---

[566] In his June 1997 affidavit, Crawford-Brown asserts the
magnitude of the difference can be explained in part by his use
of HEDR's maximum 7 day hold-up time in calculating the HEDR
doses, rather than the central value of a 3.5 day hold-up time.
Use of a 7 day hold-up time would decrease HEDR's doses even
further in comparison to Crawford-Brown's doses which assume a
3.5 year-round hold-up time. Crawford-Brown states that in the
"actual dose calculations," both he and HEDR use the 3.5 day
hold-up time. He also states that the calculations at his
deposition included a processing value ("Lproc") of .5, although
he actually used a value of .4 in the dose calculations contained
in his report. Nevertheless, Crawford-Brown acknowledges that
"some differences" remain between his doses and those of HEDR.
(Crawford-Brown 1997 Affidavit at p. 2). (See discussion infra
regarding hold-up time).

ORDER RE SUMMARY JUDGMENT-   723

deposition estimates on two grounds: 1) his reliance on Klementiev's scientifically unreliable source term estimates; and 2) his concentration and deposition estimates are the result of an unsound methodology, evidenced by violation of the "mass balance" principle.

There is simply no doubt that **to the extent** Crawford-Brown bases his dose estimates on Stewart's scientifically unreliable results, those dose estimates are likewise scientifically unreliable. Stewart's methodologically unsound work taints and infects Crawford-Brown's dose estimates. This can be likened to a chain reaction which begins with the exclusion of Klementiev's scientifically unreliable source term estimates.

Plaintiffs recognize exclusion of Stewart's work necessitates exclusion of dose estimates based on Stewart's work. **All they can do in their response brief is note that Crawford-Brown is upfront about his "conditional:"** "The doses calculated **in this report**, therefore, are conditional upon [Stewart's] airborne concentrations and rates of deposition . . . ." (Crawford-Brown November 1995 Affidavit at p. 2)(Emphasis added).

In his June 1997 Affidavit (Foulds Ex. 21), Crawford-Brown emphasizes there are "two pieces" to his "Hanford testimony:"

> The first is the conversion factor from environmental conditions (air concentration and deposition) to dose in different ages. This component is not conditional. The second component is that dose that follows from the specific environmental analysis performed by Stewart using the release terms of Klementiev. This component IS conditional upon the results of those two authors.

(Crawford-Brown 1997 Affidavit at p. 1)(Emphasis in text).

ORDER RE SUMMARY JUDGMENT-   724

Crawford-Brown acknowledges the validity of his dose
estimates, **as derived from Stewart's concentration and deposition**
estimates, is conditional upon the reliability of Stewart's work
and, in turn, Klementiev's work. Those are the dose estimates
found in Crawford-Brown's **report**. According to Crawford-Brown:

> So any dose numbers, final numbers, rads, for
> example, that appear in here [the report], are
> conditional entirely on the concentrations and
> deposition rates that Dr. Stewart provided.

(Crawford-Brown Dep. at p. 41).

However, Crawford-Brown emphasizes that his "conversion
factors" or "fruit-to-total dose ratios" are not dependent on
Stewart's concentration and deposition estimates. Indeed, the
comparison doses which Crawford-Brown calculated at his
deposition (Defendants' Ex. 22) were based on the **same** HEDR
estimate of I-131 deposited on the ground. Even so, Crawford-
Brown's doses were considerably higher than the HEDR doses.
Essentially, Crawford-Brown says that even if Stewart's and
Klementiev's results are excluded, that does not necessarily
require exclusion of his "conversion factors."

The defendants appear to concede as much. They say:

> Crawford-Brown's misplaced reliance on Stewart
> does not, however, explain the extent of the
> disagreement between his doses and those of HEDR.
> Even if one were to substitute HEDR's iodine ground
> deposition figures for Stewart's in Crawford-Brown's
> equation, one would still obtain dose estimates up to
> <u>fifteen times higher</u> than those in HEDR. An
> explanation of this alarming discrepancy requires
> additional analysis of Crawford-Brown's methodology.

(Defendants' Opening Br. at p. 8)(Emphasis in text). Defendants
go on to argue why they believe Crawford-Brown's "fruit-to-total

ORDER RE SUMMARY JUDGMENT-   725

1 dose ratios" or "conversion factors" are scientifically

2 unreliable.

3   A question arises whether plaintiffs and Crawford-Brown have

4 committed themselves to using Stewart's and Klementiev's work as

5 part of a total package, such that it is irrelevant whether or

6 not Crawford-Brown's "conversion factors" are scientifically

7 reliable. Clearly, the intent of Crawford-Brown's report was to

8 calculate doses based on Stewart's concentration and deposition

9 estimates: "The doses calculated in this report . . . are

10 conditional upon [Stewart's] airborne concentrations and rates of

11 deposition." In their brief, plaintiffs emphasize they retained

12 Crawford-Brown "to calculate general dose ranges within the

13 isopleths[567] of air transport deposition of iodine-131 in the

14 downwind study domain **as modeled by Dr. Stewart."** (Plaintiffs'

15 Response Br. at p. 3)(Emphasis added).

16   For reasons set forth below, the court finds Dr. Crawford-

17 Brown's conversion factors are not scientifically reliable.

18 Accordingly, his dose estimates, whether derived from **Stewart's**

19 concentration and deposition estimates or **HEDR's** concentration

20 and deposition estimates, must be excluded from a jury's

21 consideration.

22 //

23 //

24 //

25 //

26 ─────────────────────────

    [567] A line on a map connecting points at which a given

27 variable has a specified constant value.

28 **ORDER RE SUMMARY JUDGMENT-   726**

1   **(2) "Fruit-to-Total Dose Ratios"/"Conversion Factors"**

2   **(Ingestion Dose)**

3   **(a) Hold-up Time**

4   One of the parameters considered by both HEDR and Crawford-

5   Brown in calculating **ingestion** dose is the "hold-up" time between

6   the collection or harvesting of the fruits and vegetables and

7   ingestion of them. This parameter is considered to take into

8   account the decay of radioiodine which occurs between harvesting

9   and ingestion. Radioiodine has an eight day half-life meaning

10 that for every eight day period after the iodine settles on the

11 crop, only one half of the then-existing radioiodine will remain.

12   Crawford-Brown uses a year round "hold-up" time of 3.5 days

13 for leafy vegetables and fruits, representing the median of the

14 range reported in Snyder, et al., "Parameters Used in the

15 Environmental Pathways and Radiological Dose Modules (DESCARTES,

16 CIDER and CRD Codes) of the Hanford Environmental Dose

17 Reconstruction Integrated Codes (HEDRIC)," (May 1994)(PNWD-2023

18 HEDR Rev. 1).[568]   (Crawford-Brown November 1995 Report at p.

19 11). The range reported in Snyder 1994 is a minimum hold-up time

20 of zero days and a maximum hold-up time of seven days. (Snyder

21 1994 at p. 6.122).

22   Defendants contend Crawford-Brown fails to consider that

23 HEDR's hold-up times apply only to the **actual harvest season** for

24 the fruits and vegetables, while he would apply his 3.5 day hold-

25 up time **during every month of the year**. For leafy vegetables,

26 _____

27   [568]   Foulds Ex. 104, hereinafter "Snyder 1994."

28 **ORDER RE SUMMARY JUDGMENT-   727**

1   HEDR assumes fresh harvest months of June through September. For

2   fruit, it assumes fresh harvest months of June through October.

3   **For non-fresh harvest months, HEDR determines food crop hold-up**

4   **time by the lapse between final harvest and the date of**

5   **consumption.**   (Snyder 1994 at p. 6.123). According to

6   defendants, Crawford-Brown's application of a year round hold-up

7   time translates to an unreasonable assumption that residents of

8   the HEDR study domain ate freshly picked fruits and vegetables

9   year round, and not just during the time they were actually

10 harvested.

11   Defendants contend that since the actual harvest season is

12 only five months and most of the iodine present on fresh fruits

13 and vegetables will have decayed within one month of deposition,

14 Crawford-Brown's 3.5 day hold-up time could apply, at most,

15 during six months of the year. By applying the hold-up time for

16 the full twelve months, defendants say Crawford-Brown

17 overestimates ingestion doses by a factor of two.

18   In his 1997 affidavit, Crawford-Brown acknowledges his use

19 of hold-up times "explains one of the differences between [his]

20 analysis and that of HEDR for the non-'harvest' seasons."

21 (Crawford-Brown 1997 Affidavit at p. 3). At his deposition,

22 Crawford-Brown testified the difference could be as high as a

23 factor of 1.9. However, he added this had to be considered in

24 the context of the "very large uncertainty" of how people were

25 getting their food during the non-harvest months, including the

26 possibility of them getting their food from "non-contaminated"

27 sources "to the fact that people have growing seasons that are

28 **ORDER RE SUMMARY JUDGMENT-   728**

1 beyond the dates that are listed [by HEDR], both before June and

2 after October in their home gardens."   (Crawford-Brown Dep. at

3 pp. 136-137).

4   Crawford-Brown reiterates this in his affidavit:

5   [Defendants] deliberately . . . ignore the
             key part of my argument: that the use of hold-up
6   times from historically remote periods of time
             (three decades prior to the analysis by HEDR)
7   is not scientifically defensible, and if they
             must be incorporated in a calculation of average
8   dose, then the values are necessarily subjective.
             They also ignored the concerns which I voiced in
9   my deposition over HEDR's choice of a fresh
             harvest season where the growing season may
10   have extended beyond the assigned periods. I
             do not disagree that a hold-up time would apply;
11   I simply disagree that the historical reconstructions
             of hold-up times used in the HEDR report satisfy
12   standards of scientific validity.

13   In addition . . . the Court is interested in
             individual-specific doses, not average doses to
14   the very large populations over which the HEDR
             analysis performs an averaging in considering
15   hold-up times. As I stated in my deposition
             testimony to the defense, more reliable hold-up
16   times should be applied at a later stage of analysis
             when specific individuals are identified, placed
17   at specific locations in the concentration field,
             and their individual-specific holdup times identified.
18   Until then, given the large variability of food
             consumption patterns between individuals in a
19   population, the averaging approach by the HEDR
             analysis is not appropriate in estimating doses
20   to specific individuals.

21 (Crawford-Brown June 1997 Affidavit at p. 3).

22   HEDR's hold-up times are indeed "subjective." According to

23 Snyder 1994, food crop hold-up times were "subjectively

24 estimated."   (Snyder 1994 at p. 6.123). However, it is a

25 "subjective" assumption that at least has some basis in reality.

26 Crawford-Brown's assumption of a year round harvest strains

27 credibility. The most he can offer in defense of his assumption

28
    ORDER RE SUMMARY JUDGMENT-   729

1 is that people "have growing seasons that are beyond the dates

2 that are listed [in HEDR]."

3   Crawford-Brown does not offer any particular evidence to

4 back up that assertion, acknowledging he had not done any review

5 of the harvest times in the Hanford area and assumed HEDR's hold-

6 up times came from a poll of farmers who run large farms.

7 (Crawford-Brown Dep. at pp. 137-38).[569] Secondly, that growing

8 seasons may in some cases extend beyond the dates used by HEDR is

9 not the same as assuming a year-round growing season which

10 includes the winter months. With regard to the possibility that

11 during the non-harvest months individuals may have received their

12 food from non-contaminated sources (the "international food

13 chain"), Crawford-Brown agreed this would **decrease** his iodine

14 doses.   (Crawford-Brown Dep. at p. 106).

15   Ultimately, Crawford-Brown tries to dodge the controversy

16 altogether by contending any discussion of hold-up times should

17 be left for individual-by-individual analysis. Hold-up times

18 will certainly vary among individuals, especially during the

19 fresh harvest season (some individuals probably ate fruits and

20 vegetables the very day they were picked). Nonetheless, this

21 does not justify the dose-increasing assumption employed by

22 Crawford-Brown in calculating his **"average"** or **"representative"**

23 doses that **even during the non-harvest months**, individuals were

24 _____

   [569] In their brief, **plaintiffs' counsel** cite various
25 documents which they say show the existence of two growing
   seasons in the Hanford area- May to July and September to
26 November. (Plaintiffs' Response Br. at pp. 20-21). However,
   even that does not establish the existence of a **year-round**
27 growing season.
28
   ORDER RE SUMMARY JUDGMENT-   730

1   eating fruits and vegetables 3.5 days or less after they were

2   harvested. It simply does not make sense.

3   Plaintiffs' counsel contend Crawford-Brown's uniform 3.5 day

4   hold-up time throughout the year is warranted because the effect

5   of hold-up time is reduced by HEDR's minimal biomass value of

6   .01. "Minimal biomass values" represent the living or dormant

7   portion of the crop that exists over the year. (Snyder 1994 at

8   p. 6.33). At his deposition, Crawford-Brown testified that

9   because the "biomass" is very low in the system during the **non-**

10 **fresh harvest months**, he did not feel it necessary, unlike HEDR,

11 to determine food crop hold-up time by the time between the final

12 harvest date and the date of consumption.[570] Nonetheless,

13 Crawford-Brown testified he did not disagree with HEDR's modeling

14 approach in this regard. Indeed, he acknowledged HEDR's approach

15 provided a more "precise" estimate, and that **his** approach

16 (uniform 3.5 day hold-up time during the non-fresh harvest

17 months) caused up to a "30 percent or so change in the dose

18 calculation." In other words, Crawford-Brown's **average** doses

19 would be 30% higher than HEDR's **representative** doses. (Crawford-

20 Brown Dep. at pp. 101-102). Upon further consideration,

21 Crawford-Brown upped the figure to 50% (Id. at p. 108), and

22 ultimately concluded the maximum difference could be as much as

23

24

25

26   [570] Crawford-Brown used the 3.5 day hold-up time instead.
        HEDR's method results in longer hold-up times for the non-fresh

27 harvest months.

28 **ORDER RE SUMMARY JUDGMENT-   731**

90% (1.9 factor).[571]

Plaintiffs' counsel attempt to downplay the significance of the hold-up time parameter. They note that among six different parameters, HEDR ranked hold-up time fifth in "Relative Importance of Parameters to Ingested Iodine-131 Activity for a Child Consuming Fresh Fruit in 1945." (Farris 1994 at p. D.15). HEDR indicated the six parameters accounted for 90% of the uncertainty in the amount of iodine ingested via the fruit pathway, but hold-up time accounted for only 10% of the overall 90% uncertainty. Id. at p. D.14. However, **Crawford-Brown** himself does not cite these figures and this clearly is not proof of the existence of a year round growing season.[572]

Counsel also assert Crawford-Brown's year-round hold-up time is "moderated" by his assumed ingestion rates. However, there is no indication as to the extent of the "moderation" and no mention of where **Crawford-Brown** discusses "moderation" because of assumptions regarding ingestion rates.

As defendants point out, any error in calculation of the

_____

[571] At his deposition, Crawford-Brown explained that the 1.9 factor results from a comparison between HEDR's use of a 3.5 day hold-up time for the five month period of June through October (the fresh harvest months) versus his use of a 3.5 for the entire year (all 12 months). According to Crawford-Brown, "a maximum of 12 over 5" is "2.2" which is modified to "about 1.9" considering "the amount of activity in the plants decays over the harvest." Crawford-Brown testified that this "happens to be the same thing that you would get if you had a 7-day mean holdup time throughout the entire year." (Crawford-Brown Dep. at pp. 136-37).

[572] Defendants note that in Crawford-Brown's analysis, hold-up time becomes a critical parameter because it assumes fruit and leafy vegetables are harvested and eaten within a few days during the entire year.

ORDER RE SUMMARY JUDGMENT-   732

1 fruit and leafy vegetables dose skews Crawford-Brown's total

2 ingestion dose since the fruit and leafy vegetables dose is the

3 basis from which he calculates total ingestion dose (via use of

4 the "fruit-to-total dose ratio" or "conversion factor").

5

6 **(b)   Justification for Extrapolation of Fruit and Leafy**

7 **Vegetables Dose to Milk and Egg Dose**

8 Defendants argue there is no scientifically valid reason for

9 tying fruit and leafy vegetables dose to the milk/eggs dose.

10 According to defendants:

11 > . . . while Crawford-Brown purports to rely
   > upon HEDR's calculations of the milk dose, **he**
12 > does not actually use HEDR's milk dose calculations.
   > He inflates his milk dose estimates by extrapolating
13 > to the milk dose the increase that he proposes to
   > the HEDR fruit-and-leafy vegetable dose. In other
14 > words, if Crawford-Brown calculates a fruit-and-leafy
   > vegetable dose that is twice as high as HEDR's
15 > fruit-and-leafy-vegetable dose, Crawford-Brown also
   > doubles HEDR's milk dose even though he is purportedly
16 > relying on HEDR's milk dose.

17 (Defendants' Reply Br. at p. 8). Defendants contend plaintiffs

18 have not offered a legitimate explanation why Crawford-Brown's

19 increased fruit and leafy vegetables dose should be applied to

20 increase the HEDR milk dose which Crawford-Brown purports to rely

21 upon.

22 Crawford-Brown's report indeed offers no criticism of HEDR's

23 milk and egg dose methodology. He explicitly states that "for

24 this category of consumption, the methodology employed by . . .

25 HEDR . . . was used in its entirety." (Crawford-Brown November

26 1995 Report at p. 11). Nevertheless, Crawford-Brown opted to

27 **infer** milk and egg dose from **his** fruit and leafy vegetables dose.

28 **ORDER RE SUMMARY JUDGMENT-   733**

The question then is why did he not just use HEDR's milk and egg

dose.

In his June 30, 1997 **affidavit**, Crawford-Brown does not

attack any of the specific assumptions and parameters underlying

HEDR's milk and egg dose methodology. Instead he broadly

asserts:

> The reason I chose not to rely on the
> reconstruction of the milk pathway developed
> by HEDR is that it is my professional opinion
> that, while a laudable goal, such posterior
> reconstructions of food consumption patterns,
> highly spatially localized, are too unreliable
> to form the basis of scientifically valid dose
> estimates for INDIVIDUALS in the area surrounding
> Hanford. More than three decades have passed
> since the most important period of time for
> iodine exposures, and historical reconstructions
> of food sources on the spatial scale are highly
> questionable.
>
> I do not believe the HEDR Project was able to
> reliably develop geographically-specific dose
> conversion factors for milk distribution patterns,
> and for this reason I averaged over the various
> geographic regions reported in the HEDR analysis
> (removing what I believe, in my professional opinion,
> to be an inappropriate degree of spatial and temporal
> accuracy in the HEDR analysis).

(Crawford-Brown June 1997 Affidavit at p. 1)(Emphasis in text).

Plaintiffs follow up on this in their brief, contending

Crawford-Brown chose to independently calculate doses via

inhalation and **direct ingestion** of leafy vegetables and fruit

because it was his expert opinion that "less uncertainty" was

associated with the direct ingestion pathway than with **indirect**

ingestion of milk and eggs, which required an elaborate

reconstruction of the complex milk distribution system.

According to plaintiffs:

ORDER RE SUMMARY JUDGMENT-   734

> Just as EPA models do not require a comprehensive
> investigation and calculation for each pathway for
> multiple pathway exposures, <u>Daubert</u> does not require
> plaintiffs' experts to comprehensively investigate
> every aspect of the HEDR model in order to critically
> review the **source term and air transport components.**
> **The cost of analyzing the milk distribution system,**
> **which is necessary to provide an independent**
> **calculation of the milk dose is simply prohibitive,**
> **<u>and is not the central area of dispute between the</u>**
> **<u>parties on dose</u>.** Unlike the HEDR Source Term and
> RATCHET models which relies in large part on historical
> data supplied by the defendants, HEDR's milk pathway
> model depends on data substantially gathered from
> independent sources- therefore plaintiffs did not
> allocate the substantial resources required to
> reconstruct the milk distribution system, which
> HEDR had already attempted to a limited extent. With
> the exception of consumption rates which will be
> furnished in the next phase from the individual
> plaintiffs, **Crawford-Brown accepted HEDR's method**
> **for calculating the <u>average</u> ingestion dose for the**
> **dairy pathway, and using their analysis reasonably**
> **inferred the <u>average</u> doses for the dairy pathway from**
> **his direct calculation of the leafy vegetables and**
> **fruit dose.**

(Plaintiffs' Response Br. at pp. 7-8)(Emphasis added).

Elsewhere, plaintiffs acknowledge Crawford-Brown has

"inferred a range of average individual total doses which relies

on HEDR's analysis of the milk pathway." Plaintiffs say this

provides them "with a means to compare the effects on average

doses that result from differences in **parameters and inputs in**

the source term and air transport components, on which there are

fundamental disagreements between HEDR and plaintiffs."

(Plaintiffs' Response Br. at p. 14)(Emphasis added).

The plaintiffs seem to indicate the area of dispute does **not**

pertain to the dose components of HEDR (DESCARTES[573] and

---

[573] Dynamic Estimates of Concentrations and Accumulated
Radionuclides in Terrestrial Environments.

ORDER RE SUMMARY JUDGMENT-   735

CIDER[574]), but rather the **source term and air transport components** (STRM and RATCHET).[575] If that is the case, defendants legitimately ask why Crawford-Brown did not just run the DESCARTES and CIDER models with Klementiev's source term estimate and Stewart's concentration and deposition estimates?

Asked about this at his deposition, Crawford-Brown stated DESCARTES and CIDER were not linked up with easily accessible tools like Excel software so he developed his own software that was "simply more accessible." (Crawford-Brown Dep. at pp. 44-45). Asked whether he had any criticism of the DESCARTES and CIDER models other than the fact they were not as "accessible," Crawford-Brown stated he used a **"slightly different"** approach regarding several different parameters for inhalation and ingestion dose, but that these were **"minor points"** and HEDR had "done a proper job of developing these equations." (Id. at pp. 45-47).

The utility of Crawford-Brown's analysis (and his computation of **average doses**) is further called into question by his concession that for the purpose of calculating actual **individual** doses, it will be necessary to run the entire HEDR model first before his "fruit-to-total dose ratios" or "conversion factors" can be applied. At his deposition,

---

[574] Calculation of Individual Doses from Environmental Radionuclides.

[575] Plaintiffs assert Crawford-Brown's methodology for estimating average doses is scientifically reliable where it is based on the "HEDR dose analysis (ie, limited to its DESCARTES and CIDER models)." (Plaintiffs' Response Br. at p. 4).

ORDER RE SUMMARY JUDGMENT-   736

1 Crawford-Brown testified:

2   . . . I have to adjust my figures to [individuals']
          particular consumption patterns in the same way
3   that the HEDR group will have to adjust their
          figures. **So what would really happen would be**
4   **the HEDR methodology would be used to reestablish**
          these ratio of doses for the ingestion of . . .
5   **fruits, leafy vegetables, milk, eggs and so on, and**
          then my method of conversion factor could be applied.
6
(Crawford-Brown Dep. at p. 154)(Emphasis added).
7
8   Currently, HEDR is capable of calculating I-131 doses for

categories of "representative" (hypothetical) individuals. Just
9
as HEDR will need to tailor its analysis to specific individuals,
10
Crawford-Brown says he will need to do so as well. Thus, **after**
11
HEDR figures out the percentage contribution of each exposure
12
pathway **for a specific individual**, Crawford-Brown says he will
13
examine those percentages to determine the "fruit-to-total" dose
14
ratio **for that specific individual.**
15
          The question is: If the HEDR model is run with regard to a
16
specific individual and doses are produced for the various
17
exposure pathways based on the particular consumption information
18
supplied by the individual, why is a conversion factor necessary?
19
When the individual consumption information is supplied, arguably
20
there is no longer any need to **infer** milk and egg dose from the
21
fruit and leafy vegetables dose. In their brief, plaintiffs
22
acknowledge that "based on the specific information supplied by
23
the individual [] there will be less need for extrapolations or
24
subjectively assumed values for such parameters as produce holdup
25
time, source of dairy products, and consumption rates."
26
(Plaintiffs' Response Br. at p. 5).
27
28
ORDER RE SUMMARY JUDGMENT-   737

1   However, plaintiffs assert HEDR's "best estimate **may very**

2 **likely** depend on the ratios presented in Table 4.4 [Farris 1994]

3 to extrapolate a dose for the other multiple pathways."

4 (Plaintiffs' Response Br. at p. 16) (Emphasis added). According

5 to plaintiffs, an example of the propriety of extrapolating total

6 ingestion dose from the "fruit and leafy vegetables" dose in

7 calculating the dose for an actual individual would be if that

8 individual has a detailed recollection of his fruit and leafy

9 vegetable consumption patterns, but no such recollection about

10 his dairy consumption patterns.[576] Nevertheless, that still

11 does not take care of the issue of why HEDR's milk and egg dose

12 methodology, a methodology which Crawford-Brown is willing to

13 embrace in its "entirety," should not be used for directly

14 calculating milk and egg dose, as opposed to inferring it from a

15 "fruit and leafy vegetables" dose.

16   The bottom line is that Crawford-Brown's methodology and the

17 **average** doses produced by it, adds nothing at this stage of the

18 litigation in terms of how **individual** doses should be calculated.

19 Crawford-Brown's methodology is not an alternative to HEDR.

20   Plaintiffs assert the generic causation phase of the

21 litigation imposes no requirement on Crawford-Brown to calculate

22 **individual** doses. Plaintiffs say this is so because "the general

23 causation question . . . is whether there is sufficient

24 scientific evidence of an association between [I-131] in the

25 _____

26   [576] Presumably, there would be no need for the ratio if the
individual **did** have detailed recollection of his dairy
27 consumption patterns.
28

ORDER RE SUMMARY JUDGMENT-   738

1 **range of doses** estimated by plaintiffs' experts and the various

2 health effects claimed by plaintiffs."

3 The court has determined that is **not** the applicable

4 causation question. What is necessary is a model which is

5 scientifically reliable for the purpose of calculating **individual**

6 **doses** to determine whether an individual has received a dose in

7 excess of the "doubling dose." The "doubling dose" standard is

8 necessary for inferring whether Hanford emissions are a "more

9 likely than not" cause of the individual's disease.

10 Crawford-Brown proposes to apply his "conversion factors"

11 after the HEDR model has been run with regard to a specific

12 individual and the ratios of the doses from the various pathways

13 are "re-established." Yet, he fails to offer a compelling

14 scientific reason that his method of **inferring** milk/egg dose from

15 fruit and leafy vegetable dose is necessary when: 1) individual

16 consumption information should be available to fill in the

17 blanks; and 2) **he (Crawford-Brown)** does not say why HEDR's

18 milk/egg dose methodology is deficient for calculating milk/egg

19 doses, other than making a general assertion that it involves too

20 much uncertainty.

21 In their response brief, **plaintiffs' counsel** undertake a

22 particularized attack of HEDR's milk dose methodology (feed-to-

23 milk transfer factors, surface water consumed by cows, etc.),

24 claiming it underestimates milk doses. (Plaintiffs' Response Br.

25 at pp. 32-38; 42-43). Such an attack is not found in either

26 Crawford-Brown's expert report or in his affidavit. This is no

27 surprise since Crawford-Brown stated in his report that he was

28 **ORDER RE SUMMARY JUDGMENT-**   **739**

1 relying on HEDR's milk/egg dose methodology in its "entirety."

2 The court will not address counsel's arguments regarding HEDR's

3 milk dose methodology.

4

5 **(c)   Use of Constant Ratio to Generate <u>Average</u> Doses**

6   Defendants assert Crawford-Brown's **average** doses, whatever

7 value they may have, are not reliable because they are based on

8 an erroneous and scientifically unreliable "constant ratio"

9 assumption.

10   Crawford-Brown assumes a "constant ratio" between the fruit

11 and leafy vegetables contribution to dose and the milk and eggs

12 contribution to dose:

13   The key here, the central assumption, is that
                when you change the release term and, therefore,
14   the airborne concentrations, and, therefore,
                the deposition rates onto the ground surface,
15   at a particular cell, a particular grid block, you
                change the dose that's delivered, but you don't
16   change the relative magnitude of the component
                from eggs/milk versus the component from fruit/
17   leafy vegetables.

18 (Crawford-Brown Dep. at p. 72).

19   Defendants contend any variance in the ratio has a

20 significant impact on dose estimates. Thus, if a Richland

21 resident receives 10% of his total ingestion dose from fruit and

22 leafy vegetables and his fruit and leafy vegetables ingestion

23 dose is 10 rads, his total dose will be 100 rads (10 rad fruit

24 dose x 10.0 "fruit-to-total dose ratio"). For a comparable

25 Eltopia resident whose fruit and leafy vegetables dose is also 10

26 rads, but which constitutes 50% of his total ingestion dose, his

27 total ingestion dose is 20 rads (10 rad fruit dose x 2.0 "fruit-

28 **ORDER RE SUMMARY JUDGMENT-   740**

1 to-total dose ratio"= 20 rads). Although the Richland and

2 Eltopia resident have the same fruit and leafy vegetable dose (10

3 rads), the difference in ratios means the Eltopia resident's

4 total dose is five times less than that of the Richland

5 resident.[577]

6   Crawford-Brown acknowledged that for any particular

7 geographical cell or grid node[578], his "correction factor"

8 (conversion factor) could be high or low relative to what is

9 calculated using HEDR's entire methodology, including the milk

10 and egg distribution system. According to Crawford-Brown, some

11 "grid blocks" would be elevated by 20% and some would be too low

12 by 20%. (Crawford-Brown Dep. at p. 74).

13   Defendants assert the difference is actually well in excess

14 of 20%. They compare "fruit-to-total dose ratios" between a male

15 less than one year old residing in Richland and a male less than

16 one year old residing in Eltopia. Under HEDR's Regime 4 (milk

17 from a backyard cow fed stored feed), Crawford-Brown's "fruit-to-

18 total dose" ratio for Richland is 1.6, while for Eltopia it is

19 2.1, a 133% increase according to defendants.[579] Under HEDR's

20 _____

21 [577] Another example cited by defendants is if an individual
   drinks no milk, but lives in an area where Crawford-Brown applies
22 a "fruit-to-total dose ratio" of 8.0, the individual still ends
   up with a total ingestion dose of 80 rads even though his fruit
23 dose is only 10 rads and comprises nearly **all** of the individual's
   dose (10 rads x 8.0 equals 80 rads).

24
   [578] There are 2,091 cells or nodes within the 75,000 square
25 mile HEDR study domain.

26 [579] 2.1/1.6 = 1.3125, which by the court's calculation is a
   31% increase. Whether the increase is 133% or 31%, it still
27 exceeds Crawford-Brown's self-imposed 20% reliability test.
28
   ORDER RE SUMMARY JUDGMENT-   741

1   commercial milk consumption regime, Crawford-Brown's "fruit-to-

2   total dose ratio" for a Richland male less than 1 year old is

3   1.8, while the Eltopia ratio is 7.5, a 415% increase according to

4   defendants.[580] (See Defendants' Ex. 136 computing "fruit-to-

5   total dose ratios" based on Table 4.4 of Farris 1994).

6   Plaintiffs assert that because Crawford-Brown averages his

7   ratios over the two towns (Richland and Eltopia), "his dose

8   estimates will not differ from HEDR's by as much as '133%.'"

9   Crawford-Brown agrees the ratios as between the two towns

10 (Richland and Eltopia) vary, but asserts his ratios "are averages

11 over the two towns" which "do NOT vary." (Crawford-Brown June

12 1997 Affidavit at p. 4.)(Emphasis in text).

13   It is true, as indicated above, that Crawford-Brown combines

14 the ratios of Richland and Eltopia to arrive at an average ratio.

15 According to Crawford-Brown:

16   . . . I do not believe individual-specific ratios
                can be developed reliably at this time, so I
17   disagree with the HEDR approach of stating different
                ratios for INDIVIDUALS in these towns. That is why
18   I average over towns. The degree of variability
                between the ratios in the two towns [Richland and
19   Eltopia] . . . is meaningless and is apparently
                raised as a red herring. The issue they raise may
20   be that they believe the ratio varies systematically
                as one moves outwards from the source or swings through
21   different directions from the source. **The concern
                may be that the ratio determined from these two towns
22   is not representative of other towns. I see no
                evidence given for the claim of any systematic pattern
23   of differences in dose ratios with spatial location
                and have based my calculations on the assumption that
24   there is random variation of the dose ratios between
                the different grid blocks in the analysis.**

25 _____

26   [580] 7.5/1.8= 4.167 which by the court's calculation is a
    317% increase. Whether the increase is 415% or 317%, it greatly
27 exceeds 20%.

28
    ORDER RE SUMMARY JUDGMENT-   742

1 (Id.) (Emphasis added).

2   Although plaintiffs dispute the extent of the difference

3 between Crawford-Brown's **average** doses and HEDR's

4 "representative" doses as claimed by the defendants, neither

5 plaintiffs or Crawford-Brown make any effort to show that the

6 differences actually fall within Crawford-Brown's self-imposed

7 20% range. Plaintiffs conclusorily assert it is "not obvious"

8 that "greater ratio variations" exist within the HEDR study

9 domain. Crawford-Brown's response to the "concern" that the

10 ratio determined for Richland and Eltopia is not representative

11 of other towns is merely that he sees no evidence of any

12 systematic pattern of differences in dose ratios. Indeed, he

13 assumes there is in fact a **random variation** between the different

14 grid blocks.

15   Two of the defendants' experts, John A. Auxier and John R.

16 Frazier, prepared a comparison of Crawford-Brown's "fruit-to-

17 total dose ratios" for locations within the HEDR study domain to

18 the actual ratios for those locations. (Auxier/Frazier

19 Affidavit; Defendants' Ex. 194). Auxier has a Ph.D. in nuclear

20 engineering with over forty years of experience in health

21 physics. Frazier has a Ph.D. in physics with an emphasis in

22 health physics and approximately 20 years of experience,

23 primarily in environmental dose assessments, internal dosimetry,

24 bioassay, external radiation dosimetry, environmental sampling

25 and analysis, and radiation detection and measurement.

26   Auxier and Frazier tested Crawford-Brown's "assumption" that

27 his method would not introduce a difference, high or low, of more

28 **ORDER RE SUMMARY JUDGMENT-   743**

than 20 percent. Auxier and Frazier calculated the 1945 HEDR

doses for each of HEDR's ten exposure pathways, for both males

and females, for six age groups[581], for three different feed

regimes[582], and for seventeen cities and towns located within

the HEDR study area[583]. From these doses[584], they calculated

what Crawford-Brown's "fruit-to-total dose ratios" would be for

each of those locations. These ratios are found in Tables 2

through 7 of the Auxier/Frazier affidavit. Those ratios were

then compared with the ratios Crawford-Brown published in his

November 1995 report (the average ratios he calculated for

Richland and Eltopia **combined**).[585]

---

[581] 0, 1, 5, 10, 15 and 20

[582] Milk from cows fed on fresh pasture; milk from cows fed on stored feed; consumption of grocery (processed) milk.

[583] Baker, LaGrande, The Dalles and Pendleton, OR; Moscow, Coeur d'Alene and Lewiston, ID; Walla Walla, Pasco, Richland, Eltopia, Ellensburg, Yakima, Moses Lake, Ritzville, Wenatchee and Spokane, WA.

[584] It appears that in calculating these doses, Auxier and Frazier went strictly by HEDR and did not use Crawford-Brown's **increased** fruit and leafy vegetable doses. According to Auxier and Frazier, "the most important reason that [Crawford-Brown's] calculated doses from ingestion of fruit and leafy vegetables are higher than the doses calculated by using the HEDR method for the same pathway is that Dr. Crawford-Brown ignored the seasonal nature of consumption of fresh fruits and leafy vegetables." Auxier and Frazier do not consider this to be a scientifically reliable method since it "is less temporally detailed than the more rigorous HEDR method." (Auxier/Frazier Affidavit at p. 4).

[585] Crawford-Brown's "fruit-to-total dose ratios" or "conversion factors" are found on p. 12 of his November 1995 Report. For Regime 1 (cow fed on fresh pasture), the conversion factor is 7.8 for individuals less than 1 year old; 14.9 for individuals ages 1-4; 16.9 for individuals ages 5 to 9; 16.1 for individuals ages 10-14; 16.7 for individuals ages 15-19 years; and 12.2 for individuals ages 19 and over. For Regime 4 (cow fed on stored feed), the conversion factor is 1.7 for individuals

ORDER RE SUMMARY JUDGMENT-   744

1   Thus, for example, on Table 2, "Pathway Dose Ratios for

2   Females during 1945, Assuming Their Primary Milk Source was a

3   Backyard Cow Eating Fresh Pasture," one can see that for an

4   infant less than 1 year old residing in **Wenatchee, WA**, the

5   "fruit-to-total dose ratio" is 2.7. This is compared to

6   Crawford-Brown's "fruit-to-total dose ratio" of 7.8 for

7   individuals less than 1 year old, which he derived from his

8   examination of the percentage contributions to dose for Richland

9   and Eltopia as found in Table 4.4 of Farris 1994. The percentage

10 difference between Crawford-Brown's 7.8 ratio and the 2.7 ratio

11 for Wenatchee is 189% (7.8/2.7=2.88 or 189%). The Wenatchee

12 ratio is 189% less. For a 1 year old infant residing in

13 Wenatchee, the ratio is 4.5. Compared to Crawford-Brown's ratio

14 of 14.9 for individuals between 1 and 4 years old, the difference

15 is 232% (14.9/4.5=3.311 or 232%). The Wenatchee ratio is 232%

16 less.

17   In each of these particular examples, Crawford-Brown's

18 method significantly overestimates the **total ingestion doses**

19 received by a female infant less than 1 year old, and a 1 year

20 old female, who resided in Wenatchee in 1945 and consumed milk

21 from a backyard cow grazed on pasture. Under Crawford-Brown's

22 method the total ingestion dose is figured by extrapolating from

23 the fruit and leafy vegetables dose. Therefore, if the ratio of

24 fruit and leafy vegetable dose to total dose is less, the total

25 _____

    less than 1 year old; 2.7 for individuals ages 1-4; 2.9 for
26 individuals ages 5 to 9; 2.9 for individuals ages 10-14; 2.9 for
    individuals ages 15-19 years; and 2.3 for individuals ages 19 and
27 over.
28
    ORDER RE SUMMARY JUDGMENT-   745

dose is less. Conversely, if the ratio of fruit and leafy

vegetable dose to total dose is more, the total dose is more.

Under Crawford-Brown's method, a 2.7 ratio produces a smaller

total ingestion dose than a 7.8 ratio. A 4.5 ratio produces a

smaller total ingestion dose than a 14.9 ratio. It is simply a

matter of proportionality.[586]

Crawford-Brown derives his ratios based on doses received by

individuals in Richland and Eltopia for a single year: **1945.**

For each of the seventeen cities and towns mentioned above,

Auxier and Frazier calculated HEDR ingestion doses for the years

1945 through 1957 for a five year old who drank milk from a cow

fed on fresh pasture, and a five year old who drank milk from a

cow fed on stored feed. Auxier and Frazier also calculated HEDR

ingestion doses for the years 1945 through 1957 for an adult who

drank milk from a cow fed on fresh pasture, and an adult who

drank milk from a cow fed stored feed. From these doses, Auxier

and Frazier calculated "fruit-to-total dose ratios." The ratios

are found in Tables 8 to 11 included as part of their affidavit.

Auxier and Frazier conclude the ratios show a large

variation over **time.** They note that for a child born in Richland

and Eltopia on January 1, 1940[587] who drank milk from a backyard

cow fed on pasture grass, the ratios range from 12 to 47 during a

---

[586] Auxier and Frazier also calculated ratios for the
commercial milk consumption regime. These are found in their
Tables 6 and 7. Crawford-Brown, however, does not provide
"fruit-to-total dose ratios" for the commercial milk consumption
regime. Therefore, it is not apparent how a comparison can be
made here.

[587] This child would have been 5 years old in 1945.

ORDER RE SUMMARY JUDGMENT-   **746**

1 13 year period. See Table 8, "Pathway Dose Ratios for a

2 Hypothetical Child Born on 1/1/40, Assuming Their Primary Milk

3 Source was a Backyard Cow Eating Fresh Pasture." Crawford-

4 Brown's ratio for a child between 1 and 4 years old is 16.9.

5 This is the average he derived from the ratio for Richland (16)

6 and Eltopia (18) in 1945 as reflected on Auxier and Frazier's

7 Table 8.

8 For 1952, when the Richland individual is 12 years old,

9 Auxier and Frazier calculate a "fruit-to-total dose ratio" of 47.

10 Crawford-Brown's ratio for individuals ages 10-14 is 16.1. By

11 the court's calculation, a ratio of 47 is a 192% increase over

12 Crawford-Brown's ratio of 16.1 (47/16.1=2.92). For 1950, when

13 the Eltopia individual is 10 years old, Auxier and Frazier

14 calculate a "fruit-to-total dose ratio" of 12. By the court's

15 calculation, a ratio of 12 is a 34% decrease over Crawford-

16 Brown's ratio of 16.1 (16.1/12=1.34).

17 A review of Tables 2 through 5 and 8 through 11 reveals

18 numerous instances where Crawford-Brown's ratio is too high or

19 too low in excess of 20%. It is abundantly clear that Crawford-

20 Brown's ratios, published in his November 1995 report, do not

21 meet his own test of reliability. The court must agree with

22 Auxier and Frazier's assessment:

23   Dr. Crawford-Brown's method of calculating dose
             from ingestion of milk is predicated on his
24   claim that the ratio of the combined doses
             from ingestion of milk, eggs, fruit and vegetables
25   and the sum of the doses of ingestion of fruit
             and leafy vegetables will vary only slightly
26   as time passes and from place to place. As we
             have shown, this assertion is not valid and the
27   model built on it will not be adequate for the

28 **ORDER RE SUMMARY JUDGMENT-   747**

task of detailed dose reconstruction for specific
individuals in the HEDR study area.

(Auxier/Frazier Affidavit at p. 7).

The ratios presented in **Crawford-Brown's November 1995
report** are of no value. Even he admits as much, saying the
ratios will need to be reestablished after information is
obtained regarding **specific individuals**. However, Crawford-Brown
and the plaintiffs have offered no specific scientifically valid
reason why at the individual causation stage, the specific
individual's total ingestion dose should be **inferred** from his/her
fruit and leafy vegetable dose.

(3)   Miscellaneous Arguments by Plaintiffs

Plaintiffs' **counsel** attempt to divert attention from
deficiencies in Crawford-Brown's method. For example, they
attack **HEDR's** use of "backcasting" ratios in **its** dose estimates.
(Plaintiffs' Response Br. at pp. 24-26). They note HEDR used
national food consumption data rather than local food consumption
data and "backcasted" the national data from 1977-78 to estimate
the type and amount of food consumption for the period between
1945 and 1957. Anderson, et al.,"Estimation of 1945 to 1957 Food
Consumption," (PNWD-2113 HEDR)(March 1993)[588], p. 2.5.

Plaintiffs cite a portion of Anderson 1993 which states:

> The backcasting method seems to consistently
> underestimate rural consumption. The 1950,
> 1954, and 1969 rural studies . . . consistently
> estimated per capita egg consumption over 50
> percent higher on average than the backcasting

---

[588]   Foulds' Ex. 3.

ORDER RE SUMMARY JUDGMENT-   748

1
2

 method. On average, per capita milk consumption
 was backcast over 15 percent lower than the
 rural studies. . . .

3
4
5
6

 The backcasting method's inability to predict
 rural consumption could be offset by applying a
 rural adjustment factor, especially to obtain a
 more reliable estimate of milk. That factor should
 probably be at least 15 percent to fully offset
 underestimation of rural milk consumption.

(Anderson 1993 at p. B.11).

7
8
9
10
11
12
13
14
15
16
17

 Based on the foregoing, plaintiffs assert defendants have

failed to inform the court "that the dose estimates they rely on

for the dominant milk pathway contains a bias that remains

uncorrected." According to plaintiffs, because it is necessary

to correct the underestimate in milk and egg consumption for

rural areas, this "would" increase the dairy pathway

contributions reported at Table 4.4 in Farris 1994. In turn, say

plaintiffs, the adjusted percentage contributions "would" have

furnished Crawford-Brown with even higher dose conversion factors

than those appearing in his report.

18
19
20
21
22

 Whatever the validity of plaintiffs' argument regarding a

bias in HEDR's milk and egg doses, the fact remains that

Crawford-Brown accepted HEDR's milk and egg dose methodology in

its "entirety." Therefore, this attack on HEDR says nothing

about the reliability of **Crawford-Brown's** methodology.

23
24
25

 A section of plaintiffs' response brief (pp. 26-32) is

entitled "'Maximum' Parameter Values Apply to Court's Review of

Estimated Doses." Here, plaintiffs state as follows:

26
27
28

 . . . because defendants rely on HEDR for its
 summary judgment motions, the plaintiffs
 should be assumed to have received the <u>maximum</u>
 -not the median doses estimated by HEDR, and the

ORDER RE SUMMARY JUDGMENT- **749**

> maximum values cited in PNWD-2023 parameters
> document[589] should be employed in any calculation
> of average individual doses for summary judgment
> purposes.
>
> The parameters relied on by Crawford-Brown and
> HEDR are **not** the maximum values, which should
> dispel any suggestion that Crawford-Brown was
> intent on inflating doses. Indeed, defendant[s']
> Exhibit 136 'A'[590] does reflect that the Crawford-
> Brown and HEDR leafy vegetable and fruit ingestion
> dose parameters are comparable and reflect 'central'
> or average values, lending additional scientific
> validity to Crawford-Brown's dose estimates.
> Nonetheless, plaintiffs submit that a close inspection
> of pertinent references cited by HEDR in PNWD-2023
> warrant parameter values even higher than those used
> for Crawford-Brown's estimates and HEDR's median dose
> estimates.

(Plaintiffs' Response Br. at p. 27)(Emphasis in text).

This argument is based on plaintiffs' misperception of their generic causation burden. Plaintiffs maintain the general causation question is whether there is sufficient evidence of an association between I-131 in the **range of doses** estimated by plaintiffs' experts and the various health effects claimed by plaintiffs. That is why they had Crawford-Brown come up with a **range of doses** or "average" doses to which individuals living around Hanford may have been exposed. That is also why plaintiffs argue maximum parameter values should be employed in any calculation of **average** individual doses for summary judgment purposes.

The applicable evidentiary standard is "doubling dose." An

---

[589] Snyder, et al., 1994.

[590] Defendants' Ex. 136 is divided into an "Exhibit A-Crawford-Brown's Ingestion Dose Parameters" and an "Exhibit B-Crawford-Brown's Fruit-To-Total Dose Ratios Based on Farris 1994 Table 4.4."

ORDER RE SUMMARY JUDGMENT-   750

1   individual must prove he/she was exposed to a dose of iodine in

2   excess of the "doubling dose." Only then can it can be inferred

3   Hanford radiation is "more likely than not" a cause of the

4   individual's condition such that a jury should be allowed to

5   consider his/her case.

6   Crawford-Brown's **average** doses are of no assistance in

7   answering this question. What is needed now is a scientifically

8   reliable method for calculating **individual** doses for specific

9   individuals so it can be determined whether those specific

10 individuals were exposed to a dose of I-131 in excess of the

11 applicable "doubling dose."[591]

12   Secondly, as plaintiffs acknowledge, even Crawford-Brown

13 himself does not use HEDR's maximum parameter values. Plaintiffs

14 admit the fruit and leafy vegetable dose parameters used by

15 Crawford-Brown and HEDR are "comparable" and reflect "average"

16 values. Because Crawford-Brown does not assert that use of

17 maximum parameter values is appropriate, the plaintiffs have no

18 expert proof supporting such a position for the purpose of

19 computing either **average** doses or **individual** doses. Instead, it

20 is **plaintiffs' counsel** who argue "pertinent references" from HEDR

21 "warrant parameter values even higher than those used for

22 Crawford-Brown's estimates and HEDR's median dose estimates" and

23 discuss three parameters in particular: ingestion dose factor

24 ("DFing"); fraction of I-131 deposited onto plants ("fv"); and

25 _____

26   [591] It would seem that calculation of individual doses can
    only take place after completion of individual causation
27 discovery.

28
ORDER RE SUMMARY JUDGMENT-   751

1   percentage of iodine that remains on fruit and vegetables after

2   washing or processing.

3   Plaintiffs' counsel attack the reliability of HEDR's dose

4   estimates for a variety of reasons which are not discussed in any

5   of Crawford-Brown's materials (i.e. performance of HEDR models in

6   the VAMP "Scenario CB" exercise; alleged failure to consider

7   direct human ingestion of contaminated drinking water; rainsplash

8   rate; goat milk consumption, etc.) Whatever the validity of

9   these arguments, they do not remedy the deficiencies in Crawford-

10 Brown's methodology.

11

12   **(4)   Daubert Criteria**

13   Plaintiffs apparently suggest Crawford-Brown's dose

14 estimation analysis grows naturally and directly out of research

15 he has conducted independent of this litigation. An example,

16 according to them, is that Crawford-Brown conducted an

17 epidemiological study of occupational exposures to Hanford

18 workers which provided him with knowledge of the Hanford site and

19 familiarity with the operations and facilities which produced the

20 iodine releases. Plaintiffs note Crawford-Brown has "reviewed

21 data on the occupational exposures to Hanford workers, which is

22 independent of the work he is performing for plaintiffs."

23   Elsewhere, plaintiffs contend Crawford-Brown's dose

24 estimates "flow from a source of information specific to Hanford

25 that he reviewed and from which he obtained scientific knowledge

26 that was independent of this litigation." Plaintiffs refer to

27 the 1991 master's thesis of Sandra F. Shindle, "Thyroid Cancer

28 **ORDER RE SUMMARY JUDGMENT-   752**

1 Risk Assessment for the 1945-47 Population in the Hanford

2 Region,"[592] which was approved by Crawford-Brown in 1992 in his

3 capacity as a supervising faculty member.   Shindle, who

4 apparently later took the married name of Snyder, was the lead

5 author of the HEDR Parameters Report, referred to above as Snyder

6 1994.

7   These arguments completely ignore Crawford-Brown's

8 acknowledgement that the purpose of his analysis was to make HEDR

9 more "accessible" to plaintiffs' counsel.   According to Crawford-

10 Brown:

11   My spreadsheet was designed to allow the
              plaintiff's (sic) team to examine the influence
12   of assumptions which differ from those adopted
              by the HEDR analysis.   The defense is ignoring
13   the fact that the HEDR analysis is viewed by
              many outside the DOE system as a product of DOE,
14   precisely the organization under scrutiny in
              these lawsuits.   The plaintiff's (sic) had a
15   legitimate reason to want a model vetted by
              an outside expert (myself), where they could
16   know not only which assumptions I placed into
              the model, but could discuss with me the
17   assumptions I have not selected (and determine
              the impact of those assumptions on the final
18   dose estimates if later research revealed those
              assumptions to be valid).

19
   (Crawford-Brown 1997 Affidavit at p. 6).   Clearly, Crawford-
20
   Brown's work does not grow naturally and directly out of research
21
   he has conducted independent of this litigation.
22
        Crawford-Brown acknowledges that his work for plaintiffs has
23
   not been peer-reviewed.   He contends "such risk analyses are
24
   routine and not appropriate for publication in the scientific
25
   literature to which I normally submit my papers for publication."
26
   _____

27   [592] Foulds' Ex. 99.

28 **ORDER RE SUMMARY JUDGMENT-   753**

1 (Id. at p. 5).   Nonetheless, the lack of peer review does nothing

2 to bolster the reliability of Crawford-Brown's work.

3   Plaintiffs and Crawford-Brown argue it is a generally

4 accepted standard methodology to calculate risk based on a

5 particular pathway of exposure even when there are multiple

6 pathways of exposure.   Even if that is true in the **abstract**,

7 there is no compelling indication that what Crawford-Brown

8 **specifically** did in this case comports with standard methodology

9 or has been (or would be) generally accepted within the

10 scientific community.

11   Finally, as is evident from the discussion above, Crawford-

12 Brown's "fruit-to-total dose ratios" or "conversion factors" are

13 subject to a significant rate of error.

14   An evaluation of the Daubert criteria confirms the

15 unreliability of Crawford-Brown's analysis for calculating either

16 average or individual doses.

17

18   **d.   Fit/Relevancy**

19   Crawford-Brown's **average** I-131 doses clearly are of no

20 assistance in determining whether a particular individual

21 received a dose of iodine in excess of the applicable "doubling

22 dose" I-131 related health conditions.   While his method is

23 potentially relevant for calculation of **individual** I-131 doses,

24 he has not justified its reliability (or its necessity) for

25 calculating such doses.

26 **//**

27 **//**

28
ORDER RE SUMMARY JUDGMENT-   754

e.   Conclusion

The **average** doses found in Crawford-Brown's 1995 **report** are not scientifically reliable because they are derived from Dr. Stewart's scientifically unreliable airborne concentration and deposition estimates which, in turn, are derived from Dr. Klementiev's scientifically unreliable source term estimates.

Secondly, and as a separate matter, Crawford-Brown's **average** doses are based on two methodologically unsound assumptions: 1) a year-round hold-up time of 3.5 days for the purpose of computing the fruit and leafy vegetables component of the total average dose; and 2) that the ratio of fruit and leafy vegetable dose to milk/egg dose will remain constant throughout all geographical locations and all years of exposure within the HEDR study domain.

Crawford-Brown's **average** doses are of no assistance to the trier of fact in determining whether a particular individual received a dose of iodine in excess of the applicable "doubling dose" for I-131 related health conditions. Plaintiffs mistakenly believe they need to produce evidence of **average** iodine doses in order to meet what they think to be their generic causation burden of proof. In addition to excluding Crawford-Brown's **average** doses on the basis of <u>Daubert's</u> reliability prong (Prong 1), the court will exclude those doses on the basis of the fitness prong (Prong 2).

While Crawford-Brown maintains his ratio methodology can be used to calculate specific **individual** doses at the appropriate time, he has not advanced a specific scientifically valid reason

ORDER RE SUMMARY JUDGMENT-   755

1 why total individual dose should be **inferred** from fruit and leafy

2 vegetable doses as opposed to using the **entire** HEDR dose

3 methodology, including its milk/egg dose methodology, based on

4 the food consumption information supplied by the particular

5 individual. This should not be construed as a finding that

6 HEDR's dose methodology is necessarily reliable for that purpose.

7 Rather, it is merely a recognition that the HEDR model is

8 Crawford-Brown's standard of reference. While plaintiffs may

9 wish to have Crawford-Brown on hand at trial for the purpose of

10 establishing individual doses, Crawford-Brown has not

11 demonstrated that his participation would assist the trier of

12 fact.

13   For all the foregoing reasons, Crawford-Brown will be

14 excluded from testifying at trial regarding the iodine dose

15 estimation method contained in his November 1995 report.[593]

16 //

17 //

18 _____

19

20   [593] Crawford-Brown apparently also provided plaintiffs' with
a plutonium dose estimation method. Defendants do not launch a
21 <u>Daubert</u> attack against that method or any other plutonium dose
estimation method supplied by plaintiffs. The reason is obvious.
For the most part, plaintiffs' average plutonium doses, based on
22 the maximizing assumptions presented in their Table II do not
exceed the applicable "doubling dose."
23   The doubling doses for thyroid cancer are in general much
smaller than those for the non-thyroid cancers. Furthermore,
24 because of the amount of I-131 released, even by HEDR estimates,
there is significantly greater potential that individuals
25 received I-131 doses in excess of the applicable doubling doses
for thyroid cancer. Indeed, Frazier's cumulative I-131 dose to
26 the thyroid (derived from Goble's method) for an adult individual
residing in Ringold during the entire release period is 435,833
27 millirem (436 rem).

28 **ORDER RE SUMMARY JUDGMENT-   756**

VII.   SUMMARY JUDGMENT/RULE 54(b) CERTIFICATION

After its exhaustive review of the scientific evidence in this case, the court fully understands why <u>Daubert</u> requires judicial officers to assume the role of "gatekeeper." The complexity of the evidence in this case, indeed the mere appearance of complexity and the manipulation of numbers in some instances, could easily inspire the most astute jury to reach an erroneous conclusion that exposure to Hanford emissions was a cause in fact of an individual's disease.

What remains of plaintiffs' scientific evidence, and granting them all favorable inferences therefrom, establishes the legal viability of claims based on the following health conditions: 1) Thyroid Cancer (including thyroid nodules and adenomas); 2) Non-autoimmune clinical and subclinical hypothyroidism; 3) Bone Cancer; 4) Lung Cancer; 5) Salivary Gland Cancer; and 6) Breast Cancer (Lactating Female). All claims premised on health conditions other than these are **DISMISSED with prejudice.**

Thyroid cancer claims (including claims for thyroid nodules and adenomas) cannot proceed to trial unless there is proof of I-131 exposure in excess of the following doubling doses: 5 rads for those 0 to 4 at the time of exposure; 10 rads for those 5 to 9 at the time of exposure; 33 rads for those 10 to 19 at the time of exposure; and 100 rads for those 20 and over at the time of exposure. All thyroid cancer claims (including claims for thyroid nodules and adenomas) based on exposures equivalent to or less than these doses are **DISMISSED with prejudice.**

ORDER RE SUMMARY JUDGMENT-   757

1   Clinical (non-autoimmune) hypothyroidism claims cannot

2   proceed to trial unless there is proof of I-131 exposure in

3   excess of 750 rads. Subclinical (non-autoimmune) hypothyroidism

4   claims cannot proceed to trial unless there is proof of exposure

5   in excess of 350 rads. All non-autoimmune clinical

6   hypothyroidism and subclinical hypothyroidism claims based on

7   exposures equivalent to or less than these doses are **DISMISSED**

8   **with prejudice.**

9   Furthermore, the thyroid cancer or non-autoimmune

10   hypothyroidism claim of any individual whose I-131 exposure

11   occurred solely after **1960** is **DISMISSED with prejudice.**

12   Salivary gland cancer claims cannot proceed to trial unless

13   there is proof of radiation exposure (I-131 and any non-iodine

14   exposure) in excess of the following doubling doses: 33 rem for

15   adults (ages 20 and over) at the time of exposure; 17 rem for

16   children (ages 10-19) at the time of exposure; and 10 rem for

17   infants (ages 0-9) at the time of exposure. All salivary gland

18   cancer claims based on exposures equivalent to or less than these

19   doses are **DISMISSED with prejudice.** Any such claims based solely

20   on I-131 exposure occurring after 1960 are likewise **DISMISSED**

21   **with prejudice.**

22   Breast cancer claims (lactating females only) cannot proceed

23   to trial unless there is proof of radiation exposure (I-131 and

24   any non-iodine exposure) in excess of 63 rem during lactation

25   periods. All such claims based on exposures equivalent to or

26   less than this dose are **DISMISSED with prejudice.** Any such

27   claims based solely on I-131 exposure occurring after 1960 are

28
ORDER RE SUMMARY JUDGMENT-   758

1 likewise **DISMISSED with prejudice.**

2   Bone cancer claims cannot proceed to trial unless those

3 claims are asserted by plaintiffs who lived in Ringold

4 continuously from 1944 to 1987 and were exposed to non-iodine

5 radiation in excess of 167 rem. All other bone cancer claims are

6 **DISMISSED with prejudice.**

7   Lung cancer claims cannot proceed to trial unless asserted

8 by individuals who: a) resided continuously in Ringold from 1944

9 to 1987; b) are non-smokers; c) were exposed to non-iodine

10 radiation at age 10 or less and d) the exposure is in excess of

11 77 rem. All other lung cancer claims are **DISMISSED with**

12 **prejudice.**

13   Plaintiffs who assert emotional distress claims based on

14 mere exposure to radiation must prove exposure in excess of at

15 least one of the doubling doses set forth above (with regard to

16 thyroid cancer, non-autoimmune hypothyroidism, bone cancer, lung

17 cancer, salivary gland cancer and breast cancer in a lactating

18 female), and otherwise satisfy the criteria specified above (i.e.

19 resided in Ringold continuously between 1944 and 1987). In the

20 absence of such exposure, fear of contracting a physical

21 condition is not reasonable because there is not the requisite

22 level of increased risk.

23   For the purpose of calculating I-131 dose to any individual,

24 the reports of Dr. Goble, Dr. Cochran, Dr. Klementiev, Dr.

25 Stewart and Dr. Crawford-Brown, identified above, shall not be

26 considered. For the purpose of calculating non-iodine dose to

27 any individual, the reports of Dr. Klementiev and Dr. Hattis,

28 **ORDER RE SUMMARY JUDGMENT-   759**

1 identified above, shall not be considered.

2   Accordingly, **IT IS HEREBY ORDERED:**

3   1) Defendants' Motion for Summary Judgment (Ct. Rec. 902 and

4 904) re I-131 health effects is **GRANTED** to the extent set forth

5 above.

6   2) Defendants' Motion in Limine re Lawrence Mayer (Ct. Rec.

7 906a) is **GRANTED.**

8   3) Defendants' Motion in Limine re Edward Radford (Ct. Rec.

9 906b) is **GRANTED in part** and **DENIED in part** as set forth above.

10   4) Defendants' Motion in Limine re A. James Ruttenber (Ct.

11 Rec. 906c) is **GRANTED.**

12   5) Defendants' Motion in Limine re Thomas Cochran (Ct. Rec.

13 906d) is **GRANTED.**

14   6) Defendants' Motion in Limine re Robert Goble (Ct. Rec.

15 906e) is **GRANTED.**

16   7) Defendants' Motion in Limine re Sara Peters and Douglas

17 Gnepp (Ct. Rec. 907a) is **GRANTED.**

18   8) Defendants' Motion in Limine re Richard Clapp and the R-

19 11 Survey (Ct. Rec. 907b) is **GRANTED.**

20   9) Defendants' Motion in Limine re Viktor Ivanov (Ct. Rec.

21 907c) is **GRANTED.**

22   10) Defendants' Motion in Limine re Alexandre Klementiev (I-

23 131 Source Term Analysis) (Ct. Rec. 907d) is **GRANTED.**

24   11) Defendants' Motion in Limine re Douglas Stewart (Ct. Rec.

25 907e) is **GRANTED.**

26   12) Defendants' Motion in Limine re Douglas Crawford-Brown

27 (Ct. Rec. 907f) is **GRANTED.**

28

ORDER RE SUMMARY JUDGMENT-   **760**

13) Defendants' Motion re River Emissions (Ct. Rec. 930 and 932) is **GRANTED** such that all claims based on hexavalent chromium exposure are **DISMISSED with prejudice** and the reports of Dale Hattis and Sidney Katz are **excluded**.

14) Defendants' Motion re Non-Iodine Air Pathway Emissions (Ct. Rec. 930 and 933) is **GRANTED** to the extent set forth above.

15) Defendants' Motion in Limine re Alexandre Klementiev (Plutonium Source Term Analysis) (Ct. Rec. 1007) is **GRANTED.**

16) Plaintiffs' Motions to Strike portions of defendants' various reply briefs (Ct. Rec. 1073, 1077, 1111 and 1175) are **DENIED** or rendered **MOOT** to the extent indicated in this order.

17) Plaintiffs' Motion for Certification (Ct. Rec. 1125) is **DENIED.**

18) Plaintiffs' motion for oral argument (Ct. Rec. 1193) is **DENIED.**

19) Various motions to exceed page limitations (Ct. Rec. 1104, 1119 and 1143) and to extend time (Ct. Rec. 1070, 1103, 1142, 1153 and 1171) are **GRANTED.**
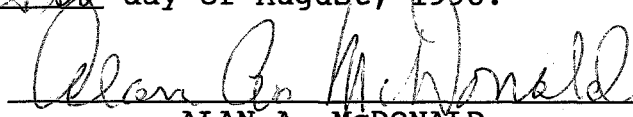
20) Various motions to expedite hearing (Ct. Rec. 1075, 1078 and 1108) are **DENIED** as being **MOOT.**

THE CLERK OF THE COURT SHALL ENTER JUDGMENT FOR THE DEFENDANTS AND AGAINST THE PLAINTIFFS AS SET FORTH HEREIN. PURSUANT TO FED. R. CIV. P. 54(b), THIS IS A FINAL JUDGMENT BECAUSE IT INVOLVES ULTIMATE DISPOSITION OF CLAIMS IN THE COURSE OF A MULTIPLE CLAIMS ACTION. THE COURT FINDS THERE IS NO JUST REASON TO DELAY ENTRY OF FINAL JUDGMENT AS TO THOSE CLAIMS WHICH ARE HEREIN DISMISSED WITH PREJUDICE. APPELLATE RESOLUTION IS

ORDER RE SUMMARY JUDGMENT-   761

1 APPROPRIATE NOW FOR THE PURPOSE OF DETERMINING WHICH CLAIMS

2 SHOULD PROCEED INTO PHASE III INDIVIDUAL CAUSATION DISCOVERY AND

3 EVENTUALLY TO TRIAL. THE APPELLATE COURT WILL NOT BE REQUIRED TO

4 ADDRESS SIMILAR LEGAL OR FACTUAL ISSUES REGARDING THE CLAIMS

5 STILL PENDING BEFORE THIS COURT IN THIS LITIGATION. ACCORDINGLY,

6 AN APPEAL LIES FROM THIS ORDER. FED. R. CIV. P. 54(a).

7   IT IS SO ORDERED. The Clerk of the Court shall forward

8 copies of this order and the judgment to liaison counsel for

9 plaintiffs and defendants.

10   DATED this ___ day of August, 1998.

11

12   ALAN A. McDONALD
                      Senior United States District Judge

13

14

15

16

17

18

19

20

21

22

23

24

25

26

27

28

ORDER RE SUMMARY JUDGMENT-   762